ACOUSTIC-REFLEX DYNAMICS AND TEMPORARY THRESHOLD SHIFT

BY

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It has been postulated that the acoustic reflex (AR) protects people's hearing from the deleterious effects of intense noise. Individuals vary widely in the characteristics of this reflex in response to the same sound stimulus. Wide variability has also been noted in temporary threshold shifts (TTS) caused by high intensity noise exposure. TTS variability may be due, in part, to individual differences in the protective function of the AR.

This study used AR parameters to explain the variation in TTS. The following experimental question was asked: Do correlations exist between TTS caused by a two-hour exposure to broadband noise and acoustic reflex magnitude, decay, threshold or latency?

The broadband noise exposure produced significant behavioral threshold shift at 1.0, 2.0, 3.0, 4.0 and 6.0 kHz. TTS did not occur at 0.5 kHz. Subjects with the most sensitive hearing suffered the greatest TTS. These results indicated that as behavioral thresholds increased within the normal range, TTS decreased. Test results indicated a relationship between ear canal volume and the frequency at which maximum TTS occurred. Subjects with greatest TTS at 3.0 kHz had ear canal volumes significantly larger than those subjects with greatest TTS at 6.0 kHz.

Statistically significant negative correlations were found between AR magnitude elicited by broadband noise and TTS. The more vigorous the AR, the less the resultant TTS due to broadband noise exposure. AR decay was also correlated to TTS such that subjects with excessive AR decay suffered high TTS. AR threshold and latency did not correlate to TTS. Multiple regression modeling that involved all significantly related variables improved TTS predictability over simple linear regression modeling.

These data suggest that the AR influences TTS development and AR properties are useful in TTS

prediction. Additional research is needed to quantify this relation and examine the AR influence for different noise exposures.

CHAPTER I
BACKGROUND AND PURPOSE

Introduction

Exposure to high intensity noise can cause numerous temporary and permanent auditory system changes. The evidence is overwhelming. A relationship between loud noise and hearing loss has been accepted for generations, and volumes of research have centered on noise-induced hearing loss as the most obvious resultant of hazardous noise exposure.

Exposure to loud sound for a short period will cause a reversible drop in hearing sensitivity commonly called a temporary threshold shift (TTS). Since Urbantschitsch first discovered TTS in 1881, the phenomenon has received considerable attention in the literature (Ward, 1973). One question that has been addressed without resolution is: Why does TTS vary so widely among individuals exposed to an identical noise stimulus? People are not homogeneously affected by exposure to the same noise (Burns, 1973). One consistent observation in TTS studies is the considerable subject-to-subject variability

(Melnick, 1974). The amount of TTS an individual suffers depends upon two independent factors--the physical properties of the noise and specific qualities of the individual's auditory system. A better understanding of the second set of factors may provide useful information about the effects of noise on hearing.

Spectral characteristics, intensity, duration and the temporal pattern of noise are physical properties of the signal directly related to TTS and have been defined in considerable detail. Auditory system features related to TTS can be classified into three broad groups: 1. static characteristics of the middle ear which include mass, geometry, elasticity, size, etc; 2. dynamic characteristics of the middle-ear muscles such as latency, threshold, magnitude and fatigue; and, 3. inner ear characteristics such as stiffness of the cochlear partition, density and spacing of hair cells, thickness of the tectorial membrane, rate of oxygen utilization, etc. (Ward, 1965). Non-auditory correlates of TTS (eg. eye color, sex, smoking history, vascular and cardiovascular condition) have also been found and could account for, in part, some of this variability. These non-auditory correlates are less clearly defined and represent an important area of continued research.

This study investigated the middle-ear reflex as one possible source of variability in temporary hearing loss.

Stimuli that produce TTS also produce middle-ear reflex activity. When these muscles are contracted, the transmission of sound energy via the middle-ear cavity is reduced. The extent of attenuation is dependent upon the dynamic properties of the acoustic reflex (AR). This reduced energy transfer (in certain acoustic situations) to the sensory structures of the organ of Corti changes as middle-ear muscle reflex threshold, duration and contractile strength varies in each individual. Researchers have reported wide variability in individual data regarding these properties of the human AR response (Borg and Odman, 1979; Borg, 1980; Gerhardt and Hepler, 1982). Due to the AR's effect on energy transmission, it would seem logical that this variability in AR would also be a factor in the variability of TTS.

The AR is strongly influenced by its dynamic properties. The time between onset of a sound stimulus and an AR response is called latency. Magnitude of AR contraction varies with intensity and has a graded influence on sound transmission. Another AR property is reflex fatigue or decay. During continuous stimulation the magnitude of the AR begins to decline. The fairly rapid fatigue noted in AR contraction has been the major argument against its having any useful protective influence (Tonndorf,1976). However, recently it has been found that the human acoustic reflex is active throughout exposure to industrial noise (Borg, Nilsson and Liden, 1979) and, in fact, may provide a very credible protective function for the auditory system.

Karlovich, Luterman and Abbs (1972), through the use of a dichotic paradigm (exposing noise in one ear and reflex eliciting stimuli in the other ear), showed a reduction in the amount of TTS that developed in the presence of reflex eliciting tones in the contralateral ear. Using a similar procedure Karlovich and Wiley (1974) observed that reduction in TTS was dependent upon the temporal characteristics of the reflex elicitor. These researchers hypothesized that TTS which developed in their subjects was influenced by the AR.

If the AR influences TTS development, then a relation should exist between properties of the AR and TTS; however, many inconsistencies can be found in the literature about this possible relationship. Most of these studies were accomplished using short-term exposures (minutes). For example, Coles (1969) found no significant relation between AR and TTS; however, Johansson, Kylin and Langfy (1967) identified "close correlations" between TTS and dynamic properties of the AR. In the industrial setting, TTS usually develops from work-related noise exposures during the course of the workday. The efficacy of inferring the relation between AR and TTS during long-term exposures (hours) from short-term exposure (minutes) studies is questionable. In one study using lengthy noise-exposure durations, Gerhardt and Hepler (1983) noted a significant correlation between magnitude of AR contraction and TTS. Other research using lengthy noise exposure suggests that the middle-ear reflex may play an important role in predicting TTS (Borg and Odman, 1979; Gerhardt, Melnick and Ferraro, 1979).

Research on the AR/TTS relationship using both short- and long-term noise exposures suggests the AR as a source of variability in TTS that is induced by hazardous noise exposure. A systematic investigation of this relation during long-term exposure is needed and could result in a test measure useful in predicting TTS.

The research objective of this study was to answer the following question: Do correlations exist between TTS caused by a two-hour exposure to broadband noise and acoustic reflex magnitude, decay, threshold or latency? Research is needed that will assist in developing procedures which predict susceptibility to high intensity noise. To date, the connection between TTS and permanent threshold shift (PTS) has not been firmly established; however, research continues. The AR/TTS relationship, when finally understood, could prove extremely useful in formulating a susceptibility test battery for noise-induced PTS.

In 1830 Fosbroke observed everyone was not equally susceptible to the deleterious effects of noise (Ward, 1980a). A hundred years later, Peyser performed probably the first test designed to measure an individual's susceptibility to noise-induced hearing loss (Ward, 1965). This test was based upon the hypothesis that those people most likely to experience TTS from short periods of exposure to loud sound also would be most susceptible to noise induced hearing loss (NIHL). Ward (1965) hypothesized the best index of PTS is the TTS obtained from the noise the person encounters on his job. TTS continues to be the most popular response variable used in studying noise susceptibility in humans. While the connection between TTS and PTS remains unclear, it seems intuitive that one exists. This relation is more apparent in studies reporting group rather than individual data. Presently, the strongest index of the hazards of noise is TTS. The ability to reliably predict TTS could represent a first step toward a valid susceptibility index for NIHL.

Review of the Literature

Temporary Threshold Shift

Excessive noise exposure creates changes within the auditory system with its most obvious effect on hearing sensitivity. The difference in hearing threshold level measured before and after exposure to noise is defined as a threshold shift. If this shift in sensitivity recovers within minutes, hours, or even days after exposure, it is called a temporary threshold shift (TTS) or auditory fatigue. If recovery does not occur, the loss is classified as a permanent threshold shift (PTS).

J.J. Muller (1871) was probably the first researcher to induce auditory fatigue (Ward, 1973). He caused TTS in one of his ears with a tuning fork (frequency N). Then, while comparing the sound of a second tuning fork (frequency N/2) between his exposed and unexposed ears, he noticed a difference in tone quality.

Victor Urbantschitsch (1881) suggested this change was actually a shift in absolute hearing sensitivity in the exposed ear (Ward, 1973). After creating a TTS in one ear, he used a similar testing protocol but attempted to obtain an estimate of hearing threshold. His tuning fork was heard for "several seconds" in the unexposed ear while it was undetected in the exposed ear. Urbantschitsch also demonstrated that this loss of hearing sensitivity was frequency specific and lasted only for a very brief time.

Since this early beginning a considerable amount of research has been devoted toward understanding and defining this phenomenon. In these studies two major terms are generally used to describe the temporal nature of reversible changes in hearing sensitivity. "Adaptation" refers to a temporary shift in hearing sensitivity occurring during the first three minutes of sound exposure and has been observed for all sound intensities. Recovery of this loss is usually complete within one to two minutes after exposure ends. "Auditory fatigue," on the other hand, is a longer lasting shift in hearing sensitivity that is generally proportional to the intensity and duration of the fatiguing stimulus (Elliott and Fraser, 1970).

Thwing (1955) investigated the spatial distribution of adaptation and found that the maximum response to continuous sound stimuli occurred at the adapting frequency with a symmetrical distribution around that point. When a pulsed stimulus was used, adaptation was greatly reduced and recovery was more rapid. Researchers have found the spatial characteristics of auditory fatigue entirely different from adaptation. Dishoeck (1948) noted maximum TTS occurred at progressively higher frequencies as intensity of the exposured increased. Typically, the frequency exihibiting the greatest shift in hearing sensitivity occurred one-half to one octave above stimulus frequency (Dishoeck, 1948; Hood, Poole and Freedman, 1976).

When observing shifts in hearing sensitivity, it is at times extremely difficult to differentiate between TTS due to adaptation and TTS caused by auditory fatigue. This is especially true at high intensity exposure levels. Recovery from these exposures tends to exhibit a diphasic recovery pattern (Hirsh and Ward, 1952). Within the first two minutes post-exposure, there is a rapid recovery from TTS followed by a short-term increase ("bounce") in TTS (figure 1). This reversal is complete by two minutes post-exposure (Elliott and Fraser, 1970) and the rest of the recovery follows an exponential course (Ward, 1973). TTS measured during the first two minutes of this recovery period is thought to reflect both adaptation and fatigue (Ward, 1973). To measure auditory fatigue without the confounding influence of adaptation, testing must be accomplished after delay of two minutes. TTS_2 (the measurement of threshold shift two minutes post-exposure) is widely accepted as a uniform reference for auditory fatigue and is commonly used to refer to the TTS resulting from noise exposure (Melnick, 1979).

The TTS a person suffers from a given noise exposure is highly variable. TTS depends upon the physical properties of the noise and the manner in which noise influences the hearing of the individual. The spectrum, intensity, duration and temporal pattern of the noise all contribute to the TTS induced by a sound. For moderate exposure levels (80 to 105 dB SPL) with durations less

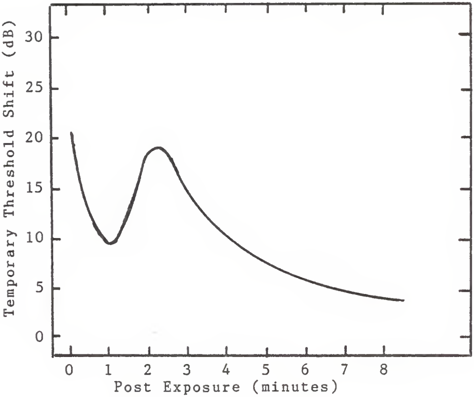

Figure 1. Diphasic recovery from temporary threshold shift (adapted from Hirsh and Ward, 1952).

than eight hours, TTS_2 shows linear growth (figure 2) with the increasing noise intensity (Melnick, 1979).

Generally, TTS growth is more rapid for high frequency than low frequency stimulation (Ward, 1973). At a comparable intensity level, the higher the exposure frequency the greater the TTS produced, at least up to about 4.0 to 6.0 kHz. TTS recovery at low frequencies tends to be slower than TTS at high frequencies. Because of this, it has been speculated that TTS at low frequencies may constitute a greater hearing hazard than comparable TTS at high frequencies (Hamernik, Henderson, Coling and Slepecky 1980).

Intensity and duration tend to trade in their effect on TTS (Melnick, 1978). The higher the intensity and the longer the exposure the greater the probability of auditory fatigue. Time and intensity must be considered together in calculating potential threat to hearing and, in fact, form the basis for PTS damage risk criteria established by various governmental agencies (U.S. Environmental Protection Agency, 1979).

The growth of TTS has an upper and lower limit (Melnick, 1978). The lower limit corresponds to an exposure level which will produce virtually no shift in behavioral sensitivity regardless of duration, nor will it interfere with TTS recovery produced by a prior exposure to a higher level. This safe exposure has been termed

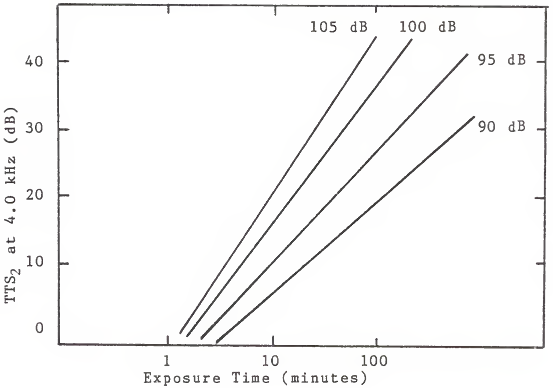

Figure 2. Temporary threshold shift at 4.0 kHz measured 2 minutes after exposure to 1700 Hz octave-band noise (adapted from Ward, Glorig and Sklar, 1959).

"effective quiet" and has been found to vary with frequency (Ward, Cushing and Burns, 1976). They reported this level to be around 76 dB for octave band exposures centered about .25 and .5 kHz and 68 dB for 1.0, 2.0 and 4.0 kHz octave band exposures. After about 8 to 12 hours of exposure, duration no longer seems to influence TTS (Melnick, 1978). TTS continues to grow to a maximum about this time and then remain constant (Melnick and Maves, 1974). Melnick (1978) uses the lower limits of TTS to estimate the expected upper limits of TTS produced from various exposures. For example, a two-hour, low-frequency, octave band exposure can be expected to create about 25 dB of TTS while an equivalent high frequency exposure will cause about 32 dB. These figures correlate well with the U.S. Environmental Protection Agency (1971) prediction of around 32 dB of TTS ("worst-case" prediction) from exposure to a 95 dB, 4.0 kHz, pure tone for two hours.

Trittipoe (1959) found that exposure to 70 dB sound pressure level (SPL) white noise had no effect on the recovery of TTS at 4.0 kHz while an 80 dB SPL exposure did. Extrapolation would seem to indicate 75 dB SPL as the level for effective quiet for broad-band exposure. Ward et al. (1976) reported that 70 dB could be considered as effective quiet for the worst of all possible spectra. If this value is used with Melnick's

(1978) TTS development chart, the expected TTS induced by a broadband stimulus at the maximally affected frequency should not exceed 32 dB.

The temporal pattern of noise exposure also determines in part the amount of TTS obtained from a given exposure. Of primary importance are the durations of noise periods (on-time), the durations of quiet periods (off-time), and the overall temporal pattern of the exposure and quiet intervals (Melnick, 1979). A much greater intensity level can be tolerated without significant TTS if the noise source is intermittent rather than continuous (Melnick, 1978). Separate risk criteria are usually established for impulse and continuous noise sources. Unfortunately discrete separation of these noise conditions rarely occurs in industrial noise settings and combinations of both noise types are encountered.

Hamernick, Henderson, Crossley and Salvi (1974) showed the possibility of a devastating synergistic effect when impulse and continuous noise sources were combined. Chinchillas exposed to innocuous continuous or impulse noise, when considered individually, received considerable auditory damage when these sources were combined. This synergistic effect has also been shown between noise and certain drugs when administered together in laboratory animals (Dayal, Kokshanian and Mitchell, 1971); however, there are no definitive data showing this interaction for TTS in human subjects (Melnick, 1978).

Histologic and Physiologic Basis for Adaptation and Fatigue

Hood (1950) suggested neural adaptation as the cause of psychophysical adaptation. This theory was based on his observation that the time course for neural and psychophysical adaptation at low or moderate stimulus intensities was basically the same. Hood, Poole and Freedman (1976) related adaptation to changes occurring in a particular receptor or group of receptors during their response to a noise stimulus. They hypothesized this physiological process was related to hair cell metabolism in that receptor rate of response declined until the metabolic energy expended in response to a sound stimulus was balanced by the energy available to the receptor. Degree of adaptation would depend upon receptor activity when maximally stimulated and, therefore, should be independent of stimulus intensity. They noted that since this process was the result of normal receptor activity, it could only be measured during stimulation and, hence, its rapid reversal after stimulus termination.

As with adaptation, the primary site of the injury produced by auditory fatigue is thought to be the organ of Corti. Hood et al. (1976) speculated that abnormal hair cell metabolism is also responsible for auditory fatigue in that reserve energy is exhausted through over-stimulation of receptor cells. They attribute auditory fatigue to the greater consumption of energy in the receptor cell with excessive stimulation than can be replaced. According to this hypothesis, the time required to replace these energy reserves represents the TTS recovery period.

Both mechanical and biochemical changes can occur in this delicate auditory region (Ward, 1980a). The degree of mechanical and biochemical disruption seems to follow the severity and permanency of the shift in behavioral sensitivity. The more pronounced the permanent loss of hearing, the greater the mechanical and biochemical damage to the cochlea. Immediate damage usually includes physical changes such as swelling of the outer hair cells and pyknosis of their nuclei (Ward, 1980a). Electrophysical changes include shifts in the linear portion of the input-output curve of the cochlear microphonic (CM) and a decrease in the maximum level of the CM (Wever and Lawrence, 1955).

Eldredge and Covell (1958) described a nine-point scale for rating the degree of damage caused by acoustic trauma. A rating of 1 or 2 means, histologically, that the organ of Corti is within normal limits. The 3 or 4 indicates moderate hair cell swelling with some displacement of nuclei along with a formation of small vacuoles in supporting cells. There may also be some displacement of the thin layer of cells (mesothelial) on the bottom of the basilar membrane. Ward (1980a) feels that damage to this point is probably reversible and represents the physiological counterpart of TTS. Ratings 5 through 9 describe increasing histologic damage to the cochlea that, for the most part, is not reversible and is related to PTS.

Biochemical changes include edema of the stria vascularis which appears within an hour after exposure and may persist for days (Duvall, Ward and Lauhala, 1974). There appears to be decreased oxygen tension in the cochlear duct (Ward, 1980a). Since neither vasoconstriction nor vasodilation seems to occur until higher exposure levels (Perlman and Kimura, 1962), this decrease in oxygen availability, at least for moderate exposure intensities, seems to be related to increased oxygen consumption rather than a change in oxygen supply (Ward, 1980a).

Histologically, outer hair cells seem to be the most vulnerable to noise exposure which induces TTS and PTS, especially in the 3.0 to 6.0 kHz region (Chung, 1980). Numerous theories have attempted to explain this vulnerability. Crow, Guild and Polvogot (1934) speculated that low blood supply in this region may contribute to an increased susceptibility. Vulnerability of the 4.0 kHz region has also been explained by fluid streaming generated through stapes movement (Hilding, 1953) and accelerations of the travelling wave (Schuknecht and Tonndorf, 1960). Ear canal resonance (around 3.0 kHz), combined with the fact that the most severe hearing loss occurs one-half to one octave higher than the center frequency of the noise exposure, may explain the weakness of this region (Tonndorf, 1976).

There may be an ordering to the hair cell damage in this region as intensity is increased (Robertson and Johnstone, 1980). In a study of acoustic trauma in guinea pigs, as intensity increased the first row of outer hair cells were damaged initially, followed by inner hair cell damage and then damage to the second and third rows of outer hair cells. There was a difference noted in the spatial distribution of inner and outer hair cell damage. Inner hair cell damage was focused in the vicinity of the exposure frequency while outer hair cell damage spread basally.

Ward and Duvall (1971) noted a histologic difference between animals exposed to noise that caused TTS and PTS. In animals with PTS there was, in addition to outer hair cell damage, a loss of inner hair cells in the first and lower second turns of the cochlea. Animals that exhibited only TTS had inner hair cells intact. There was good correlation between total exposure energy and amount of PTS and structural damage but this was not true for animals only exhibiting TTS. The noise intensity seemed more critical than exposure duration.

Relation between PTS and TTS

In 1930, Peyser suggested using TTS to predict susceptibility to permanent damage from high intensity sound (Ward, 1973). Since that time, TTS has received considerable attention in the literature. Studies relating PTS to TTS in humans are based on two assumptions (Melnick, 1979): 1) the basic physiologic processes for both are similar, differing only in degree; and 2) the relations between the physical properties of the noise and the development of PTS and TTS are also similar.

It seems reasonable that these assumptions are valid and that a relation does exist between these two phenomena. Histologically, both seem to be due to tissue changes along the cochlear partition with a maximum effect in the 4.0 to 6.0 kHz region (Elliott and Fraser, 1970; Chung, 1980). These authors also note that PTS and TTS audiograms usually agree if the PTS is caused by long term exposure. If the PTS is caused by a short, chronic exposure, the maximum PTS occurs at a slightly lower frequency than TTS (Miller, Watson and Covell, 1963).

Generally, PTS and TTS grow linearly with the logarithm of time (Ward, 1965), and both measures are widely variable among individuals exposed to the same noise source (Melnick, 1978).

Numerous hypotheses regarding the relation between PTS and TTS have been offered. Ward (1973) suggested the possibility of permanent hearing loss if the TTS from one day of noise exposure failed to recover prior to the next day of work. He also noted that the TTS produced by eight hours of noise exposure can be used to infer the PTS caused by ten years of hazardous noise exposure (Ward, 1965). Further, he speculated that the best predictor of PTS may be the TTS resulting from the specific noise to which the individual is routinely exposed.

Mills, Adkins and Gilbert (1981) found that TTS reached a maximum after about eight hours of sound exposure and maintained that level regardless of duration. They supported a hypothesis advanced by Mills, Gengel, Watson and Miller (1970) that was similar to Ward's (1965): the asymptotic level of TTS produced by sound of fixed level and spectrum is related to the maximum PTS that can be produced by 10 years of exposure to noise.

Based upon the similarities between PTS and TTS, as well as these hypotheses, it would seem that TTS would be useful in predicting individual susceptibility to noise. Unfortunately, this has not proven to be the case.

Information regarding PTS is incomplete and its nature has not been fully defined. Data exist indicating the pattern and time-course for PTS induced by a steady-state noise exposure of a repeated 8 hour duration for several years, but little definitive data exist for other exposure conditions (Melnick, 1979). In the study of the PTS/TTS relation, this lack of information plus the difficulty in designing appropriate studies to test this relationship has led to very limited success in predicting PTS from TTS. The predictive power of TTS for group behavior appears good in some studies; however, it is only fair when individuals are considered.

Jerger and Carhart (1956) found a significant correlation between the TTS induced by a stimulus of 3.0 kHz, 100 dB SPL, and the PTS at 3.0 and 4.0 kHz following exposure to jet-engine noise. This relation was found for grouped data and they warned against individual prediction of PTS based on TTS because of the extreme variability in their data among subjects. Burns and Robinson (1970) also noted a relation between PTS and TTS when their data were averaged for a group comparison; however, extremely large variations in subject response led them to conclude that TTS was not a good predictor of susceptibility.

Not all researchers have found a relation between PTS and TTS. Sataloff, Vassallo and Menduke (1965) studied the relation in 33 subjects. They correlated the amount of PTS in Naval personnel following exposure to job related hazardous noise with TTS measures obtained 11 years previously. No significant relation was found. Harris (1965) also found no significant predictive relation in diesel-engine room personnel. Humes (1977) and Ward (1980b) both noted the speculative nature of any relation between PTS and TTS.

Primarily because of the failure to find a better PTS predictor, TTS continues to be the most popular response variable used in studying noise susceptibility in humans. Even in the absence of definitive data proving a PTS/TTS relation, these studies have been used to fill the gap in developing damage risk criteria which assess the risks of PTS from noise exposure (Melnick, 1979). Hopefully, future research will solve the mystery of this relationship and current indices of TTS can be related accurately to PTS.

Middle Ear Transmission

The peripheral hearing mechanism is composed of three divisions: the outer, middle and inner ear. The primary function of the middle ear is to transmit the acoustic information received by the outer ear to the inner ear. This transmission must occur with enough force so that energy flow is not reduced by the change in medium from air in the outer ear to fluid in the inner ear. The middle ear increases sound intensity about 1000 times to accomplish this impedance matching function (Yost and Nielsen, 1977). The efficient transfer of sound to the cochlea depends upon the amplification that occurs during this transmission process through the middle ear.

All frequency components of a sound stimulus are not transmitted equally through the middle ear. The inertia, elasticity and mass of the middle ear structures interact differently with various parts of the frequency spectrum. Generally, low frequencies are attenuated by elastic forces while high frequencies are altered by the mass and inertia of the system (Yost and Nielsen, 1977). The overall effect of these characteristics is to reduce signal transmission in the low and high frequencies while passing and enhancing signals in the middle frequency range.

Any interference with the effectiveness of this middle-ear transfer will be reflected in the signal reaching the inner ear. There are structures in the middle ear which can impede sound transmission. These include the two smallest striated muscles in the human body which attach to three hinged bones (ossicular chain) in the middle ear. These muscles, when activated, have the inherent capacity to exert tension with very little displacement. The tensor tympani rises from the inside cartilaginous surface of the eustachian tube, and from the bony canal which encases the muscle. It attaches to the upper end of the manubrium of the malleus (Zemlin, 1981). Contraction of this muscle increases impedance through the middle ear cleft by causing a medial and anterior pull on the manubrium.

The stapedius, the smaller of the two muscles, is completely encased in a bony chamber adjoining the facial canal on the posterior wall of the tympanic cavity. Stapedial muscle fibers arise from the walls of the canal which house the muscle and converge upon a small tendon which emerges into the tympanic cavity. This tendon attaches to a muscular process at the neck of the stapes above the posterior crus (Zemlin, 1981). Contraction of this muscle causes lateral pull on the stapes footplate, resulting in its posterior withdrawal at roughly a right angle to the direction of ossicular chain movement. This dislodging of the footplate and downward stapedial movement stiffens the ossicular chain thereby increasing overall impedance to sound transmission through the middle ear. Researchers have noted that this impedance increase may be due to more than just a stiffness change in the middle ear. The vibratory pattern of the stapes may also be altered by stapedius contraction during high-intensity sound exposure (Moller, 1960) and could effect the overall impedance by altering the coupling between the middle ear and cochlea (Moller, 1974).

Anatomically these two muscles are antagonists but physiologically they are synergists (Dallos, 1961). When stimulated to contract, the muscles exert opposing forces perpendicular to the primary rotational axis of the ossicular chain. Therefore, their simultaneous contraction will increase rigidity and stiffness of the ossicular chain resulting in the reduction of sound transmission through the middle ear system. During electrical stimulation, Moller (1965) reported attenuation due to combined contraction that was greater than the attenuation resulting from contraction of either muscle individually. Looking at the relation between sound pressure level and cochlear microphonic, he noted 2 dB more attenuation at 0.5 kHz when the stapedius and tensor tympani were stimulated together.

Acoustic Reflex

Contraction of the tensor tympani and stapedius muscles can occur in response to auditory and nonauditory stimuli. Tactile stimulation of various facial regions (eg. stroking the cheek, puffing of air into the eye, etc.) as well as somatic features such as chewing or vocalizing may elicit muscle contraction (Dallos, 1961).

Shutting the eyes tightly, swallowing and cutaneous stimulating of the external ear and auditory canal have also been shown to elicit middle-ear contraction (Zakrisson, 1974). When these muscles contract in response to loud sound, the reflex causes a feedback response which alters stimulus input to the inner ear.

The exact neural course of this reflex in man is not known but inferences can be drawn from research on rabbits (Borg, 1973). During this reflex response in rabbits, the auditory nerve carries the signal to the ventral cochlear nucleus which projects stimulus information to the medial superior olivary nucleus (Borg, 1973). The next synapse in this reflex arc is in the motor nuclei of the facial and trigeminal nerves. The feedback response is completed as these motor nuclei cause contraction of the stapedius (facial) and the tensor tympani (trigeminal) muscles bilaterally. Borg identified this reflex arc as a very secure, short-latency pathway. He also presented evidence for a slower pathway which may involve the red nucleus or reticular formation; however, little is known about this slower feedback mechanism.

This middle-ear response to loud sound has been most commonly labeled the acoustic reflex (AR), but is also referred to as the stapedial reflex, aural (interaural) reflex and middle-ear reflex. It has been reported that the human AR is largely mediated by contraction of the stapedius muscle (Jepsen, 1955), especially at intensity levels below 110 dB SPL. Some researchers have noted tensor tympani contraction only as part of a startle response to acoustic and non-acoustic stimulation (Klockhoff, 1961; Djupesland, 1967).

The effect of the AR on the auditory system is not static since its temporal features and magnitude of response are directly related to the spectrum and duration of the stimulus. The dynamic properties of the AR that interact with the sound stimulus to modify input to the cochlea include threshold, magnitude, latency and duration of the AR response.

Acoustic Reflex Threshold

Bilateral AR contraction occurs independent of stimulus presentation mode (monaural or binaural), and the threshold of this response is well above the threshold for behavioral sensitivity. AR threshold has been defined as the sound intensity necessary to elicit a measurable change in middle ear acoustic impedance (McPherson and Thompson, 1977). To increase reproducibility of AR threshold, some researchers have arbitrarily chosen a baseline impedance response and then calculated maximum impedance change (Moller, 1962; Borg, 1972). The sound intensity corresponding to 10% of maximum impedance shift has been labeled AR threshold. McPherson and Thompson report this method for determining AR threshold less reliable because the procedure requires two measurements, hence one more opportunity for error and variability.

The AR threshold in normal-hearing subjects has been found to vary with the spectral content of the elicitor (Flottorp, Djupesland and Winther, 1971; Margolis, Dubno and Wilson, 1980). As signal width is varied within a critical band, there is very little shift in AR threshold. Once the critical band is exceeded, the reflex is elicited with smaller intensities. Energy below 0.7 kHz does not appear to contribute to the sensitivity of the AR elicited by a broadband stimulus. Spectral intervals between 0.7 and 6.0 kHz contribute equally to AR threshold. That is, filtering of a broadband noise within this frequency range will produce a 4 dB/octave increase in the threshold of the AR. Therefore, the reflex thresholds for broadband stimuli will be lower than reflex thresholds for tonal stimuli.

Ipsilateral and contralateral stimulation of the AR does not result in symmetrical thresholds (Laukli and Mair, 1980). Generally, AR thresholds for pure tones presented contralaterally, and measured with an impedance bridge, lie between 75 and 95 dB hearing level (HL) for frequencies .25 to 4.0 kHz (Metz, 1952; Jepsen, 1963). The average value is about 85 dB HL. Sensitivity is greater for ipsilateral stimulation (Moller, 1962,1961) and this relation has been noted over the entire dynamic range of the AR. The ipsilateral AR threshold has been reported, on the average, 5 to 8 dB more sensitive than the contralaterally stimulated AR (Zakrisson, 1974). Bilateral presentation of the stimulus is approximately 3 dB more efficient than ipsilateral stimulation (Moller, 1974). AR thresholds in response to a broadband noise have been reported around 70 dB SPL (Dallos, 1964; Jerger, Jerger and Mauldin, 1972; Kaplan, Gilman and Dirks, 1977).

Jerger, Jerger and Mauldin (1972) examined AR threshold as a function of age, and noted that intensity needed to elicit AR threshold (in dB SPL) decreased with increasing age. In addition, no significant sex bias was noted in their study. Osterhammel and Osterhammel (1979) found that AR thresholds (in dB HL) for frequencies 0.5, 1.0 and 2.0 kHz did not differ significantly as a function of age and sex in adult age groups. Only in younger age groups (ages 10 to 12) was age a significant factor in AR threshold. In this study, when AR thresholds at 0.5, 1.0 and 2.0 kHz were expressed in dB SL, the threshold decreased with increasing age as reported by Jerger et al. (1972). This decrease amounted to about 3.5 dB per decade. Silverman, Silman and Miller (1983) found that age related to AR threshold elicited by broadband noise but not by pure tones. In their study changes in AR thresholds associated with the aging process began in the fourth age-decade and were more pronounced when thresholds were measured in 1 dB as opposed to 5 dB increments. Younger decades formed one homogeneous group while older adults formed another. The mean difference between the 20 to 29 year group and the 60-69 year group was 11.1 dB (1 dB measurement condition). The lack of any significant correlation between age and AR threshold for pure tone elicitors supports the results of Osterhammel and Osterhammel.

The threshold of AR contraction to sound stimuli decreases as duration of the stimulus increases (Djupesland, Sundby and Flottorp, 1973). Djupesland and Zwislocki (1971) noted a 25 dB decrease in reflex threshold as duration of a 2.0 kHz stimulating tone increased from 10 to 1000 msec. Woodford, Henderson and Hamernik (1975) found variations in AR threshold as much as 30 dB over a 10 to 500 msec range of stimulus durations. Djupesland et al. (1973) reported, for stimulus durations shorter than 50 to 80 msec, the relation between sound intensity and stimulus duration to be about the same for all stimuli (.25, 0.5, 1.0, 2.0, 4.0 kHz and white noise). They speculated that this temporal summation of the AR is independent of the type of stimulus within this range of signal durations. These researchers also found that the signal duration at which the AR threshold appears to stabilize ("knee" of the intensity/duration function) varies with the frequency of the stimulus. In other words, the dramatic effect of the stimulus duration on AR threshold covers a wider range of signal durations for high frequency elicitors. Djupesland (1975) hypothesized a greater transmission loss for low frequency stimuli may be the reason for this frequency effect. The dependence of AR threshold on signal duration supports the theory that this relation is due to differences in temporal summation, and the locus of this temporal summation must be occurring at or below the superior olivary complex within the acoustic reflex arc (Djupesland and Zwislocki, 1971).

In investigating the AR, Dallos (1973) noted that the threshold function of the AR closely reflects the ear's sensitivity curve. Barry and Resnick (1976) reported that decrease in AR threshold as a function of stimulus duration corresponded to the decrease in behavioral threshold that was observed due to changes in the temporal pattern of the signal. This was found for frequencies 0.5, 1.0 and 2.0 kHz but not for 4.0 kHz. In their study, AR threshold occurred at a relatively constant sensation level above behavioral threshold independent of stimulus duration at all test frequencies but 4.0 kHz. These researchers hypothesized the mean temporal integration function for AR threshold appears to be comparable to that for behavioral thresholds at 0.5, 1.0 and 2.0 kHz. They suggested that results using a 4.0 kHz stimulus indicate the auditory system processes energy less efficiently at that frequency than at lower frequencies.

Numerous investigators have reported on the reliability of AR threshold. Weiss, Mundie, Cashin and Shinaburger (1962) found contralaterally elicited AR thresholds to be repeatable within 1 dB during the same test session and 5 dB during test sessions one week apart. Jerger et al. (1972) reported a 6.4 dB standard deviation for test-retest reliability of AR thresholds. Using ipsilaterally presented stimuli, Borg and Zakrisson (1974) reported a 5 dB standard deviation in AR thresholds. Chun and Raffin (1979) report contralateral and ipsilateral reflex thresholds equally variable with a standard deviation of 6 dB. Chermak, Dengerink, and Dengerink (1983) reported that the maximum variation of 3.6 dB in their study, although statistically significant, was not clinically significant. Gerhardt and Hepler (1983) noted variation in reflex threshold of only 1.95 dB in their subjects. The consenus of the literature concerning reflex threshold is that this response is a highly reliable and repeatable parameter of the AR.

Acoustic Reflex Magnitude

The AR has a dynamic range of 30 dB from threshold of response to saturation for pure tone stimuli (Dallos, 1964), and around 50 dB for broadband noise (Wilson and McBride, 1978). In the majority of studies magnitude of AR response is reported in percent of maximum or decibel change from a baseline AR threshold. Normalizing AR magnitude seems to reduce variability and allow for trend analysis, but somewhat limits comparisons among stimuli and investigations (Wilson and McBride, 1978). More recent studies have attempted to quantify the magnitude of AR response in terms of absolute impedance and susceptance (Block and Wightman, 1977, Wilson and McBride, 1978; Zito and Roberto, 1980). These issues will be addressed later under "Methods of AR Measurement."

AR magnitude is graded and directly related to intensity of the stimulus. This response increases with stimulus intensity at least to levels of 116 dB SPL (Wilson and McBride, 1978) and is frequency-dependent. Moller (1962) noted some interaction of signal duration and magnitude of AR response. The increase in AR amplitude with increasing intensity, elicited by a short, 25 msec tone burst, was less than the effect produced by increased signal durations.

Djupesland, Flottorp and Winther (1967) reported maximum reflex amplitude of 18 dB above AR threshold at .25 kHz and 28 dB at 1.0 and 2.0 kHz. Habener and Snyder's (1974) data indicated magnitude was greatest at 2.0 kHz and least at 4.0 kHz.

Wilson and McBride (1978) noted the frequency-dependence of AR amplitude and reported the largest reflex response to broadband noise and 1.0 kHz pure tone stimuli and smallest response to .25 and 4.0 kHz signals. They also noted the slopes (rise time--discussed previously) for pure tone elicitors were steeper than those for broadband stimuli. In this study, the researchers varied the probe tone frequency used to measure middle-ear impedance change and found that a 660 Hz probe tone reflected larger AR magnitudes than the 220 Hz probe tone.

When relating mode of presentation to AR magnitude, Moller (1962) found that binaural stimulation elicited the largest reflex magnitude followed by ipsilateral and then contralateral stimulation. These differences were even greater for 25 msec tones than tones of 500 msec. Magnitude of AR response has also been related to age with subjects 20 to 40 years old exhibiting the most consistent amplitudes at frequencies of 0.5, 1.0, 2.0 and 4.0 kHz (Jerger et al., 1972).

Acoustic Reflex Latency

The latency of any muscle contraction is usually defined as the time between appropriate stimulation and a noticeable biological response (reflex threshold). Latency of the AR is the time required for middle-ear muscle response following a sound stimulus. Any latency value will depend upon the definition of reflex threshold. There is inconsistency in the literature as to what constitutes a noticeable change in middle-ear muscle response. Moller (1972) and Borg (1972) defined latency as the time between stimulus onset and the point at which the AR reaches 10% of maximum amplitude. Colletti (1975) used the 5% point, while other researchers suggested the point of any noticeable impedance change (Metz, 1951; Moller, 1958; Sunderland, 1974; Strasser, 1975).

Regardless of the protocol for measuring AR latency, this property is reported as being inversely related to stimulus intensity (Borg, 1972; Dallos, 1964,1973). The more intense the stimulus, the shorter the initial latency.

Sunderland (1974) found latency increased as signal rise time increased. McPherson and Thompson (1977) suggest the AR may be viewed as an "energy related phenomenon." Latency of the initial AR response is related to total energy of the input signal before the reflex occurs. Since stimulus rise time affects energy available at any given instant, it will control initial latency of the AR response.

Some variations in the reported normative values of latency have been due to the measurement method, i.e., cochlear microphonic (CM), impedance, electromyography (EMG), etc. Measurements of latency using CM as an index have been reported by Galambos and Rupert (1959). At high intensity stimulus levels the latency for stapedial contraction was 15 to 20 msec. The shortest measures of AR latency in man have been reported in EMG studies where latency periods of 10 to 11 msec to pure tone and noise stimuli have been noted (Perlman and Case, 1939; Fisch and Schulthess, 1963).

Acoustic impedance has been the most frequently used clinical measure of AR activity in man. Impedance bridges are available which permit recording of both the ipsilateral and contralateral AR. Most frequently, measures of contralateral reflex parameters are used for clinical and research purposes. A sound stimulus is produced in the opposite ear which elicits the AR bilaterally. The impedance bridge records changes in the ipsilateral ear's ability to transmit a probe tone (usually 220 Hz) through the middle ear. AR contraction will alter conduction of this probe tone and the values of

AR properties will be reflected in the characteristics of middle-ear impedance change.

McPherson and Thompson (1977) noted differences in latency due to time constants of various impedance bridges. The time constant determines the speed at which the device can respond which directly affects its measurement capabilities. Normative latency values of 150 msec near threshold and 25-35 msec at high sound intensities have been reported using impedance measures (Metz, 1951; Moller, 1958). Dallos (1964) reported a normal range between 40 and 160 msec. Considerable variation in AR latency has also been noted for individual subjects and between different subjects (Borg, 1972; Dallos, 1964,1973).

In analyzing the temporal pattern of AR response, researchers have examined different latent periods for possible clinical utility. Dallos (1964) described the AR as asymetrical and compared the "on" response to the "off" response. While onset latency was found to be nonlinear and inversely proportional to intensity, the off-set latency was reported longer in duration and independent of stimulus intensity. McPherson and Thompson (1977) suggested this difference in linearity means the contraction process of the AR differs from the relaxation process and allows consideration of each as a different parameter of AR activity. Other researchers have tried to define latency parameters in order to use this measure more effectively in differential diagnosis of lesions along the AR reflex pathway (Colletti, 1974; Norris, Stelmachowicz, Bowling and Taylor, 1974; Borg, 1982). Typically, AR latency has been segmented into a measure of initial reflex onset time, a growth period to full contraction, a value that reflects response time to stimulus termination and a measure of the time required for muscle relaxation following stimulation.

Colletti (1974) defined onset latency as the time interval between stimulus onset and 5% of maximum amplitude. He called rise time the period from 10 to 90% of maximum amplitude. Off-set latency was defined as the time period between stimulus off-set and a fall to 95% of maximum amplitude. The time required to decline from 90 to 10% amplitude after stimulus termination was called decay time. Norris et al. (1974) used somewhat different definitions for roughly the same parameters. Borg (1982) defined rise time as the period between stimulus onset to a 50% maximum amplitude in his correlation of AR latency to auditory lesions in rabbits. He cautioned that latency parameters, particularly rise time, are dependent upon stimulus intensity, and care must therefore be taken in interpretation of abnormal latency responses after lesions to the reflex pathway. Regardless of their definitions of latency parameters, all of these researchers suggested that AR latency shifts provide useful information in differential diagnosis of auditory lesions.

Acoustic Reflex Decay

During continuous sound stimulation the AR adapts (Metz, 1951; Dallos, 1964; Borg and Odman, 1979), and the rate of this adaptation seems to be both intensity and frequency dependent (Dallos, 1964; Johansson et al., 1967; Holmes, 1978). Some researchers have attempted to standardize the definition of AR decay. They defined this fatiguing process as AR "half-life" which is the time required for AR amplitude to be reduced by 50% (Anderson, Barr and Wedenberg, 1970; McPherson and Thompson, 1977). Others have plotted decay as simply the return of the AR from maximum amplitude to pre-threshold baseline during continuous sound stimulation.

The exact mechanism of AR decay is not known. Anderson et al. (1970) suggested that because this action is frequency dependent, the mechanism is probably in the afferent portion of the reflex arc. Borg and Odman (1979) note that individual pairs of ipsilateral and contralateral ARs are almost identical, so the stapedius muscle itself is not the cause of the fatigue. They also feel the site of origin for this decay is in the periphery, probably the inner ear.

Generally, the rate of AR decay increases with increased stimulus frequency (Johansson et al., 1967). These researchers found almost immediate decay in response to high frequency stimulus (3.0 kHz) and very little relaxation to low-frequency tones (500 Hz). Anderson et al. (1970) investigated the "half-life" of the AR elicited by pure tones at 10 dB SL reference AR threshold. This half-life was several minutes at 0.5 kHz, 14 seconds at 2.0 kHz and 7 seconds at 4.0 kHz. Habener and Snyder (1974) also noted very little decay in response to low frequency stimuli.

Holmes (1978) found AR strength decreased rapidly during the first minute for both 0.5 and 2.0 kHz elicitors. At 0.5 kHz, the response decreased 34.7%, while at 2.0 kHz the decay was 75.7%. She noted the decay rate declined after the first minute for both frequencies, although some additional decay was noted during the rest of the eight minute tracking period. Her results showed that while magnitude of AR contraction was greater for a higher frequency stimulus, its higher rate of decay actually reduced AR effectiveness when compared to the low frequency elicitor.

The relation between frequency and AR decay seems to be clear, but how intensity relates to this relaxation process seems somewhat more ambiguous. According to Djupesland, Flottorp and Winther (1967), the duration of

AR response increases as stimulus intensity increases. Dallos (1964) demonstrated an inverse relation between decay rate and stimulus intensity. The lower the stimulus intensity the greater the decay. Kaplan, Gilman and Dirks (1976), however, found no difference in rate of decay over a 12 dB intensity range.

The AR appears to be more resistant to decay when the stimulus is broadband noise. Presumably this is because of its random nature which serves to continually re-arouse the reflex (Ward, 1973). Ward (1961) found more AR decay to pure-tone stimulation than noise stimulation. He also noted more reflex activity with low frequency noise than high frequency noise. The decay appeared to asympote after about three minutes at approximately 50% of initial AR magnitude. Ward reported the decay linear with respect to log time during the period 15 seconds to 2 minutes.

Djupesland, Flottorp and Winther (1966) recorded impedance changes in humans exposed to different octave-bands of noise 10 dB above AR threshold. He also concluded that low-frequency noise bands were more effective at maintaining the AR. In this study complete adaptation occurred within 120 seconds.

Recently, researchers have been interested in the rate of AR decay during exposures to noise more consistent with industrial settings (Borg, Nilsson and Liden, 1979; Nilsson, Borg and Liden, 1980). In these studies with bilateral reflex recordings, middle-ear reflex activity was found to be virtually unaffected, even after 5 to 7 hours exposure. These studies suggest the AR strongly influences the auditory input during industrial noise exposures.

Protective Influence of the AR

The AR is thought to provide protection for the delicate structures of the inner ear during hazardous noise exposure (Ward, 1965; Moller, 1974; Nilsson, Borg and Liden, 1980; Borg, Nilsson and Engstrom, 1983). Researchers have found that the AR can reduce the sound energy reaching the cochlea by as much as 20 dB, mainly in the low frequency range (Borg, 1968; Cancura, 1970; Dallos, 1973). This reduction in sound energy transmitted to the cochlea via the middle-ear cavity occurs primarily for stimuli below 2.0 kHz (Karlovich, Abbs and Luterman, 1972; Dallos, 1973). Maximum reduction in transmission is about 0.6 to 0.7 dB per decibel increase in stimulus intensity up to about 20 dB above the threshold of the AR (Borg, 1968; Yost and Nielsen, 1977). Since the time for AR contraction is around 10 msec for very loud stimuli and as long as 150 msec for lower intensity signals, the protection afforded is dependent upon duration of the stimulus. Short duration sounds of sudden onset are not greatly attenuated by the AR.

Ward (1963) believed there was no affect in humans on the transmission of frequencies above 2.0 kHz during middle-ear muscle contraction. Later Ward (1966) speculated that some signal enhancement may occur for high frequencies. He based this premise on differences in observed temporary threshold shift (TTS) between males and females after exposure to high and low frequency stimuli. Other investigators have failed to note a sex bias in TTS (Karlovich et al., 1972) or middle-ear impedance change (Shallop, 1967).

Cancura (1970) studied the effect of artificial tension applied to the middle-ear muscles after detachment from the ossicular chain. His studies indicated, with moderate tension applied, the main reduction in sound transmission occurred for frequencies below 1.0 kHz. However, during stronger contractions considerable reduction also occurred for higher frequencies.

The sound attenuation provided by AR is dependent upon the nature of the stimulus and the dynamic properties of the AR (Gerhardt and Hepler, 1983). This protective influence on the sensory structures of the organ of Corti changes as middle-ear muscle reflex threshold, duration and contractile strength varies with stimulus exposure or repeated exposures. Because of this reflex, the role the

AR plays in auditory fatigue has been debated for some time. Since TTS is so widely variable across exposed individuals, as are many dynamic properties of the AR, it would seem that the AR could be a factor in this variability. If contraction of the AR serves a protective function, a relation should exist between TTS and some property of this reflex.

The AR and TTS

It is known that the AR is modified by various disease states in the middle and inner ear or beyond. For example, Jerger, Jerger and Mauldin (1974) has shown that even a 5 dB air-bone gap may alter or prevent AR activity. Colletti (1975) has related modification in AR rise time to multiple sclerosis. Others have related the effects of cochlear lesions to AR characteristics. Norris et al. (1974) have shown a slower reflex relaxation time (post stimulation) in patients with cochlear hearing loss. The AR threshold for noise stimuli is raised in patients with a cochlear loss in hearing sensitivity (Niemeyer and Sesterhenn, 1974).

Borg (1982) looked specifically at the effect of noise exposure on AR activity in rabbits. He exposed rabbits unilaterally to a noise band (range 2.0 to 7.0 kHz) at 115 dB SPL for 15 to 30 minutes. After exposure he noted an elevation in AR threshold in the affected ear and a decrease in magnitude of response as compared to the control ear. In both ears, responses well above AR threshold exhibited a shorter rise time than responses near threshold.

Other researchers have attempted to model TTS as a function of variation in AR characteristics and many inconsistencies can be found in the literature concerning this possible relation. Most of these studies were accomplished using short-term exposures (minutes). Johansson, Kylin and Langfy (1967) found a moderate correlation between TTS and latency, rise time and full activation time of the AR. These researchers exposed human subjects to a band of noise (0.3 - 1.2 kHz) for 15 minutes at 110 dB SPL. TTS was measured two minutes post exposure. There was also a tendency for those subjects with the largest amount of AR fatigue to display the greatest TTS, although this finding was not supported by group data.

Research indicates there is increased auditory fatigue from low-frequency sound exposures during stapedius muscle paralysis (Zakrisson, 1974;1975). Zakrisson (1974) showed that TTS after exposure to narrowband, low-frequency noise was considerably greater in the affected than the nonaffected ear in subjects with unilateral stapedius muscle paralysis. Zakrisson (1975) exposed Bell's palsy patients to bandpass noise exposures of 0.5 and 2.0 kHz at 115 and 110 dB SPL. Exposure durations were 5 or 7 minutes at 0.5 kHz and 2 or 3 minutes at 2.0 kHz. TTS after the 0.5 kHz exposure was significantly greater during stapedius paralysis. Stapedius paralysis was not related to TTS following the 2.0 kHz exposure. Zakrisson also noted a tendency after the 0.5 kHz noise exposure, although not statistically significant, for a negative relation between AR attenuation and TTS in the nonaffected ear. This means that the ear which was more successful at attenuating low-frequency sound tended to sustain less TTS following low-frequency exposure.

In contrast, Fletcher and King (1963) presented a fatiguing stimulus (1.2 kHz, high-pass filtered sound at 110 to 114 dB SPL) to a group of stapedectomized patients for 9 to 10 minutes and noted opposite findings. No significant difference in TTS was found between this group and a control group of normal hearing subjects.

Another method used to study the effect of AR activity on TTS is to cause regular contraction of the AR with a sound signal while exposing subjects to a fatiguing stimulus. These results are compared to a control group where AR is allowed to function normally. Karlovich, Luterman and Abbs (1972) exposed 10 normal hearing adults to 110 dB SPL, 1.0 kHz and 100 dB SPL, 4 .0 kHz pure tone stimuli for three minutes. TTS was measured at 1414 and

5656 Hz. During exposure for the experimental group a pulsed wide-band noise was presented to the contralateral ear to maintain AR contraction. For the 1.0 kHz exposure there was a significant decrease in TTS in the experimental as opposed to the control group. There was no significant difference in TTS between groups for the 4.0 kHz exposure.

Holmes (1978) reported a significant negative correlation between maximum strength of the AR at 2.0 kHz and induced TTS. Twenty normal hearing males, age 18 to 30, were exposed to white noise for 10 minutes at 115 dB SPL. A measure of maximum AR contraction strength was obtained at 10 SL (re AR threshold) for 0.5 and 2.0 kHz pure tone elicitors. AR decay was plotted over an eight minute time period and expressed in terms of percent reduction in strength of reflex relative to maximum magnitude. Subjects with more vigorous middle-ear contractions to the 2.0 kHz stimulus, pre-exposure, obtained the least TTS from the high intensity broadband noise exposure. This negative relation, although not statistically significant, was also exhibited between AR response to the 0.5 kHz stimulus and TTS.

On the other hand, Brasher, Coles, Elwood and Ferres (1969) found no significant relation between TTS and initial contraction strength of the AR or contraction strength after two minutes (decay). Coles (1969) also concluded no relation between AR decay and TTS.

Simmons (1963) studied the relation between AR and noise damage in two groups of cats, one with normal middle-ear muscles and one group with demuscled ears. Each animal was exposed to a 1.0 kHz tone for two hours at an intensity of 135 dB SPL. Little correlation was found for any reflex measure of maximum attenuation or contraction and sound damage. However, this study indicated that cats with the greatest sustained AR contractions showed the least sound damage and those with the least contraction the most trauma. But, Simmons noted extreme variation in AR response within animals and believed this variability reduced the likelihood of a positive correlation between noise damage and characteristics of the AR. He believed other explanations could account for this negative relation between AR magnitude and noise trauma. Differing predispositions to auditory habituation and adaptation were noted as a possible alternative rationale. In fact, when Simmons attempted to control for these confounding variables, the relation between AR magnitude and noise damage was considerably reduced. Based upon these findings, he warned against the observation of a causal relation between individual damage susceptibility and middle-ear reflex protection. He concluded that middle-ear muscle mechanics did not seem to be a major variable in noise induced hearing loss.

In another study which failed to support a relation between AR and TTS, Turner (1974) exposed female subjects to 110 dB HL of white noise for five minutes. He measured AR magnitude at 10 dB SL for a 2.0 kHz stimulus and obtained values of absolute middle-ear impedance. No correlation was found between TTS and any of these measures.

A major argument against a protective function of the AR is the research indicating extreme AR decay during exposure to continuous sound stimuli. Recent research indicates that the AR may not fatigue equally across all noise exposures, and that various environmental conditions during the exposure may increase the effectiveness of the AR. Most notably, irregularities in the time course of industrial noise exposure may sustain contraction of the AR.

Nilsson, Borg and Liden (1980) exposed normal hearing subjects to 30 minutes of tape recorded shipyard noise at 97 dBA. Although there was tremendous variability in the adaptation of the muscle contraction across subjects, their results showed the stapedius was active throughout the exposure with only slight decay. They suggested this variability in muscle fatigue may account for some of the variability in TTS. Borg, Nilsson and Liden (1979) also concluded that in actual industrial noise invironments, fatigue of the stapedius is slight and the AR is most likely active throughout the workday.

This literature on the protective function of the AR from a short-term exposure point of view is equivocal. The utility of a protective mechanism for the auditory system lies in its ability to operate for hours in the typical industrial noise environment. Some research has centered on the effects of long duration noise exposure on the dynamic characteristics of the AR.

Gerhardt, Melnick and Farraro (1979) investigated an eight-hour, 95 dB SPL exposure to 0.5 kHz octave-band noise in chinchillas implanted with recording electrodes. The electrodes were placed on the animal's round windows and recordings of the cochlear microphonic in response to a 0.5 kHz tonal stimulus served as an index of the dynamic properties of the middle-ear muscles. Measurements of reflex threshold, magnitude of response and latency were obtained prior to the exposure, during quiet periods at 2-hour intervals throughout the exposure and after cessation of the noise. Also, the magnitude of the cochlear microphonic during the noise exposure was monitored and served as an indicator of reflex decay during the 8-hour experiment. The study indicated reflex thresholds were significantly altered as a result. On average, a 14 dB elevation in reflex thresholds was noted. By comparing the growth of TTS in chinchillas exposed to the same noise (Carder and Miller, 1972) to the reflex threshold shift identified in their animals, these researchers concluded that these two events followed the same time course. They calculated that for every 2 dB of TTS there was 1 dB of reflex threshold shift.

A second component of that study involved reflex decay. When the amplitude of the cochlear microphonic (CM) recorded during the exposure was compared to the CM amplitude at stimulus onset (before AR onset), an estimate of reflex decay was obtained. They found the response decayed approximately 50% during noise exposure (Gerhardt, Melnick and Ferraro, 1980).

Gerhardt and Hepler (1983) in a study on humans, measured middle-ear muscle activity with an electroacoustic impedance bridge. The intent of this investigation was to verify reflex threshold shifts in human subjects following exposure to noise and to relate those changes to the development and recovery of TTS. Ten subjects participated. Eight were assigned to an experimental group and the other two served as control subjects. All subjects had normal hearing and reflex thresholds. The experimental group was exposed to a 4-hour, 1.0 kHz octave-band noise in a sound field at 95 dB SPL. The control group did not receive the noise exposure. Behavioral thresholds were measured at discrete frequencies of 0.5, 1.4, 2.0 and 4.0 kHz. Contralateral AR thresholds and intensity functions were obtained to 0.5, 1.4 and 2.0 kHz pure tones with a duration of 1000 msec. The electroacoustic impedance bridge used to measure AR activity was coupled to one channel of a strip-chart recorder. Magnitude of the AR was obtained at stimulus levels of 0 to 10 dB SL, recorded in relative units of pen deflection and normalized for each subject by converting the units to a percentage of pen deflection. Measures of behavioral thresholds and reflex activity sampled prior to exposure constituted base-line values to which all subsequent measures were compared. In addition to the pre-exposure test interval, measurements were taken after 2 and 4 hours of noise exposure and represented growth periods. The final two test intervals were designated as recovery periods and occurred 1 and 24 hours after cessation of the exposure.

Test results indicated significant TTS developed at 1.4, 2.0 and 4.0 kHz but not 0.5 kHz, one octave below the center frequency of the exposure. This temporal pattern of TTS was consistent with the results found by other researchers (Melnick and Maves, 1974; Patterson, Burdick, Mozo and Camp, 1977). AR intensity functions shifted and were significantly depressed following noise exposure. Test results also showed the magnitudes of reflex response at suprathreshold levels decreased and recovered systematically with the growth and recovery periods of TTS. This decrease in magnitude was significant during all measurement periods.

Based on long-term noise exposure studies, the time course of the effects of noise exposure on AR responses is essentially the same as the course for TTS throughout growth and recovery periods. Numerous short- and long-term exposure studies relate some aspect of AR activity to the variability in noise induced TTS. This research strongly suggests a relation between TTS and properties of the AR, most notably AR threshold shift and AR magnitude.

Methods of AR Measurement

Several methods have been developed to detect and study the acoustic reflex. In animals, cochlear microphonic data have been used to document reflex activity (Gerhardt, Melnick and Ferraro, 1979). In man, "direct" measurements of the AR have involved surgical entry into the middle-ear cavity to visualize muscle contraction or to allow the placement of electrodes for electromyographic studies (McPherson and Thompson, 1977). A more practical, "indirect" approach has been to monitor change in the flow of acoustic energy at the tympanic membrane as an indication of AR activity (Wilber, 1976).

Two types of instruments have evolved in the "indirect" approach to measurement of middle-ear function (Feldman and Wilber, 1976). The electroacoustic bridge measures impedance (ie. difficulty of energy flow) and the otoadmittance meter measures admittance (ie. the ease of energy flow). The implications of measuring impedance or its reciprocal, admittance, have importance in tympanometric interpretation as an aid in the diagnosis of middle-ear pathology. However, according to Wilber (1976) the type of monitoring device used in acoustic reflex measurement is relatively unimportant. The crucial factor is the ability to detect small changes in either impedance or admittance as an indication of reflex activity.

Clinical judgments as to the presence, absence, or decay function of the AR are normally based on the visual monitoring of an impedance or admittance meter or the interpretation of an X-Y recording of AR activity. On the otoadmittance meter there are two voltage outputs which represent the two components of admittance (ie. conductance and susceptance). Susceptance represents a measure of stored energy while conductance defines the flow of energy into a resistive element. Recent investigations have utilized the conductance and

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susceptance voltages from a Grason-Stadler, Model 1720 otoadmittance meter in their analyses (Block and Wightman, 1977; Wilson and McBride, 1978; Wilson, Steckler and Jones, 1978; Block and Wiley, 1979). Comparisons of voltages prior to, during and after stimulus presentation allowed the authors to further define characteristics of the AR such as threshold, magnitude, loudness growth and adaptation.

The authors measured both components of admittance and then calculated overall admittance in the reflexive versus quiescent states. However, Block and Wightman (1977) indicated that the decision to base representations of AR magnitude on changes in total admittance or on only one admittance component was arbitrary. Wilson and McBride (1978) compared the sensitivity of the two otoadmittance outputs and concluded that there was less than 0.5 dB difference between acoustic reflex thresholds obtained with either measure. That is, within a given probe tone condition and using their signal averaging approach, the two outputs were equally sensitive. Wilson et al. (1978) found susceptance output to be more sensitive than conductance in monitoring reflex adaptation. Wiley and Block (1979) caution against expressing admittance or its reciprocal impedance solely from the measurement of one component. Both conductance and susceptance values are essential in the computations.

They also warn that in population studies means and measures of variance cannot be made with acoustic impedance data because of differences in the phase angle of resistive and reactive components. These computations must be obtained for conductance and susceptance components independently.

Noise Susceptibility

Historically, TTS following short-term exposure to noise has been used as a predictor of individual susceptibility to noise induced hearing loss. Unfortunately, the nature of the relation between TTS and PTS remains undefined. While the connection between TTS and PTS is unclear, it seems intuitive that one exists. Because of this, TTS continues to be the most popular response variable used in studying noise susceptibility in humans.

The amount of TTS obtained from a given sound exposure is highly variable and depends primarily upon the physical properties of the noise and the manner in which noise influences the hearing of an individual. The variability in auditory fatigue among people exposed to the same noise suggests some people are more "at risk" than others. Researchers have diligently sought test measures which will identify individuals who are uniquely sensitive to noise. Ward (1965) suggested that differences in static and dynamic characteristics of the middle ear, as well as variations in the inner ear, could account for gradations in susceptibility. Tests designed to assess these individual differences have been used to predict TTS (Clack and Bess, 1969; Humes, 1977; Humes, Schwartz and Bess, 1977; Humes and Bess, 1978; Gerhardt and Hepler, 1983). Some researchers have also noted that there are non-auditory characteristics that may relate to TTS (Hood, Poole and Freedman, 1976; Carter, 1980; Carlin and McCroskey, 1980; Thomas, Williams and Hoger, 1981; Dengerink, Dengerink and Chermak, 1982; Ickes and Nader, 1982). To date, each of these properties by itself has proven only a fair indicator of TTS.

One of the most compelling non-auditory observations in noise exposed individuals who suffer hearing loss has to do with eye color. For years it has been observed that blue-eyed people are more susceptible to the deleterious effects of noise than their darker-eyed counterparts. Recent research has reported strong correlations between eye color and susceptibility to TTS (Hood et al., 1976, Cunningham and Norris, 1982). Iris pigmentation is determined by a substance called melanin that also has been isolated in the stria vascularis of the cochlea. The amount of melanin in the stria vascularis can be predicted based upon the degree of iris pigmentation (Bonaccorsi, 1965). This pigment is reported to serve a protective function by aiding in cellular metabolism. It has the "ability to transport electrons not only as a biological semiconductor but also as a system for the reduction of oxidized enzymes" (Hood et al., 1976). Certain methodological differences among studies investigating the relation between eye color and TTS have resulted in conflicting findings (Karlovich, 1975; Hood et al., 1976). However, eye color appears to have some predictive value if used as part of a noise susceptibility test battery (Thomas et al., 1981).

Other research has suggested a relation between blood supply to the cochlea and TTS (Jensen, 1969; Dengerink et al., 1982; Ickes and Nader, 1982). These research efforts were based upon the hypothesis that damage caused by noise is mediated by a reduction in oxygen available to the sensory structures of the inner ear. The oxygen supply to the cochlea in human subjects has been inferred in these studies by monitoring blood pressure, digital and cephalic vasoconstriction and by increasing the oxygen content of air. Other researchers, including Ickes and Nader (1982), have suggested noise susceptibility may be correlated to stress-prone behavior because of its known effect on peripheral constriction of blood vessels. Dengerink, Dengerink and Chermak (1982) reported that TTS was negatively correlated to resting level blood pressure and vasoconstriction. They theorized constriction of peripheral blood vessels is associated with dilation and, therefore, greater blood flow in the cochlear artery. The increase in oxygen availability due to this greater blood flow serves to reduce the damaging effects of noise.

Other non-auditory correlates to TTS include sex (Ward, 1980b), smoking history (Thomas et al., 1981) and, possibly, cardiovascular condition (Dengerink et al., 1982). When included in a test battery, these non-auditory measures could significantly increase the predictive relationship between the battery and TTS.

A very promising inner ear measure of noise susceptibility is based upon the concept of non-linearity or distortion that occurs within the inner ear at moderate stimulus intensities. By-products of distortion during intense stimulation are harmonics of the stimulating signal that can be detected using special auditory techniques. The stimulus level at which the ear departs from linearity may provide an indication of the point of system breakdown which leads to TTS. It has also been found that prior noise exposure results in harmonic distortions that occur at levels lower than normal. In some people this effect is quite pronounced. Thus, investigation of harmonic distortion provides an additional way to predict the deleterious effects of noise.

The onset of this phenomenon has been measured directly by electro-physiological measurements of cochlear microphonic (CM) and indirectly through psychoacoustic methods. Lawrence and Blanchard (1954) reported an inverse relationship between onset of overload and post-exposure TTS through the use of the aural overload test in humans and CM in animals. Humes (1980) has been very successful in predicting TTS (r=-.81) through administration of this test. In his testing protocol, Humes (1978, 1980) used a modification, originally proposed by Fausti (1971), of Wegel and Lane's (1924) best beat method for determining aural overload threshold. This procedure establishes the threshold of distortion by determining the intensity level at which the distortion product of a fundamental tone interacts with a probe tone. This probe tone is strategically introduced at a frequency calculated to allow a combination effect with the distortion product. Theoretically, the lower the threshold of the fundamental tone which allows this distortion (as manifested by a beating of the signal) the more susceptible the ear to overstimulation. Numerous studies have supported the reliability of the aural overload test in predicting TTS (Lawrence and Blanchard, 1954; Clack and Bess, 1969; Humes, 1977; Humes, Schwartz and Bess, 1977; Humes and Bess, 1978).

The threshold of octave masking (TOM) test has also proven a reliable predictor of TTS (Humes, 1977). In this procedure a value is obtained for the threshold level at which a fundamental masking tone will interfere with the perception of a probe tone introduced one octave above the masker. The concept of distortion is also basic to this theory and TOM has been hypothesized as interchangeable with aural harmonic threshold in the normal ear (Clack and Bess, 1969). An easier listening task is required with the TOM test, so clinical application has been considerably broader (Humes, 1977). However, Humes (1980) notes that although a significant relation to TTS has been obtained, correlations have not been as high as with the aural overload test ($r=-.51$).

In recent years there has been renewed interest in the use of ultra-audiometric frequency (10-16 kHz) test measures to predict the ear's susceptibility to noise. Most hazardous noise exposure is broadband and thus, produces considerable displacement at the basal end of the cochlea. It is possible that shifts in behavioral threshold measures at ultra-audiometric frequencies could precede threshold shifts in lower frequency regions of the cochlea. Recent developments in the technology of earphone transducers now permit investigation of TTS in ultra-audiometric frequencies. Collins, Cullen and Berlin (1981) created TTS in the ultra-audiometric frequencies as part of a study of residual ultra-audiometric hearing. Fausti, Erickson, Frey, Rappaport and Schecter (1981) noted substantial shifts in the ultra-audiometric frequency hearing of military veterans with histories of steady-state or impulsive noise exposures. Identifying these changes and relating them to TTS in the normal audiometric range could have important future implications for the creation of a noise susceptibility test battery.

There have been other proposed tests of noise susceptibility that measure variations in inner ear characteristics. These tests have met with varying degrees of success. It would seem that any reliable test battery of noise susceptibility should include measures based upon individual differences in inner ear function.

As noted earlier, research investigating the relation between middle-ear function and TTS suggests the AR as a valid predictor of auditory fatique. Investigators have shown systematic changes in properties of the acoustic reflex as a result of hazardous noise exposure (Borg, 1982; Gerhardt and Hepler, 1983). In one study, Gerhardt and Hepler noted a significant correlation between AR magnitude pre-exposure and the TTS which developed following a four hour exposure to 1.0 kHz octave-band noise ($r=-.75$). These authors hypothesized that individuals with more vigorous AR responses were receiving more protection and consequently suffered less TTS. A systematic investigation of this relationship during long-term noise exposure could result in a valuable addition to a noise susceptibility test battery.

Some researchers have looked for precursors to TTS in an effort to identify the noise susceptible person. They have reasoned that changes could occur within the auditory system due to noise exposure prior to any behavioral shift in hearing sensitivity. They noted the audiogram many times fails to reflect the extent of anatomical disturbance within the cochlea during high intensity noise exposure. Animals exposed to noise have shown permanent cochlea hair cell damage in the absence of any behavioral changes in pure-tone sensitivity (Ward and Duvall, 1971; Hunter-Duvar and Elliott, 1972) Recent work by Lipscomb (1983) also has indicated that damage identified histologically does not always correlate with pure tone thresholds, especially in the low-frequency (apical) region of the cochlea. Rather than investigate a pre-existing condition that may render a person more susceptible to noise, some investigators have searched for test measures that are more sensitive to abnormal inner ear function than TTS. It seems reasonable that there may be other auditory measures more sensitive to inner ear noise damage than tests of behavioral threshold sensitivity.

Michael and Bienvenue (1976a) have identified subtle changes in a person's ability to discriminate speech as a possible precursor to TTS induced from noise exposure. They speculated this could occur due to widening in critical bands which may result from damage to inhibitory neurons. The outer hair cell region of the cochlear partition contains the primary efferent network and is the most susceptible area of the cochlea to noise damage. Michael and Bienvenue suggest damage here could occur independent of altering neural input to the auditory nerve and pure tone thresholds may not be affected.

Bienvenue, Violon-Singer and Michael (1977) noted that changes in the ear's frequency integration properties are often not reflected by decreased hearing sensitivity and could precede TTS during noise exposure. They proposed a test (Loudness Discrimination Index) to measure the listener's ability to detect small changes in the loudness level of various test sounds. Their test results showed that this measure may be an earlier, more sensitive indicator of noise-induced hearing change than TTS.

These abnormal behavioral findings seem to better reflect, in some cases, the condition of the cochlea than pure-tone audiometry. If future research shows a relation between individual variability in these measures and noise induced hearing loss, then these tests may represent viable options for use in a noise susceptibility test battery.

Based upon research on the relation between TTS and various non-auditory and auditory measures, an index that successfully predicts a person's temporary loss of hearing sensitivity due to sound exposure seems possible. To be most effective, the index should include measures of middle- and inner-ear weaknesses plus an ultra-audiometric frequency and/or non-auditory test measure. Through multiple regression modeling procedures, a test battery composed of measures that are sensitive to many parameters of the auditory system should prove far superior to any single test in predicting TTS. When the connection between TTS and PTS has been fully defined, this test battery could prove successful in predicting susceptibility to permanent, noise induced hearing loss.

Statement of Purpose

The loss of hearing sensitivity caused by overexposure to noise represents a serious problem in our industrialized society that adversely affects worker productivity and is one of the most costly job related disabilities. Preventive measures have centered on various administrative controls including the use of personal hearing protection and employee education.

However, even in a well administered program with informed and cooperative employees, there will be individuals uniquely susceptible to loss of hearing due to noise exposure. Identification of these "at risk" employees is essential if effective hearing conservation is to become reality for all individuals exposed to hazardous noise.

A valid and reliable index of noise susceptibility has eluded investigators for many years. One obvious reason is the difficulty encountered in designing experimental studies to test predictors of permanent hearing loss induced in humans. Retrospective studies that investigate this relation are extremely difficult to control and are fraught with many confounding variables. Researchers have looked toward predicting temporary hearing loss caused by a sound exposure as an answer to this dilemma. Of course, any value an index of TTS would have in predicting susceptibility to PTS would depend upon the relation between PTS and TTS. To date, the relation between PTS and TTS remains undefined. Researchers continue to look for successful predictors of TTS, as well as investigate the nature of the PTS/TTS relationship.

It has been postulated that contraction of the middle ear muscles serves to protect people from the deleterious effects of loud noise. Individuals vary widely in the characteristics of this reflex in response to the same sound stimulus. This individual variability has also been noted in PTS and TTS. One could reason that the variability in PTS and TTS may be, in part, due to individual variation in the protective function of the AR.

This study used the individual variability in specific parameters of the AR to predict TTS caused by a two-hour, broadband noise exposure. The following experimental question was asked:

Do correlations exist between TTS caused by a two-hour exposure to broadband noise and acoustic reflex magnitude, decay, threshold or latency?

CHAPTER II
METHODS

Subjects

This study investigated the relation between measures of acoustic reflex activity and TTS caused by a two-hour exposure to broadband noise. All subjects were between 14 and 40 years of age ($\bar{X}=25$) and were paid for their participation in the experiment. Thirty-seven subjects were selected from students and university staff members who met the following criteria: 1. normal hearing (15 dB HL re ANSI, 1969) at octave intervals from .25 through 8.0 kHz; 2. no history of chronic ear disease and a normal tympanogram; 3. no employment history which required exposure to hazardous noise for periods in excess of two years; 4. AR thresholds of less than 110 dB SPL at 1.0 kHz; 5. ability to perform a standard Bekesy procedure (modified method of adjustment) defined by repeatable thresholds (within 3 dB) at 1.0 kHz over three trial periods with stable tracings for up to one minute; 6. Bekesy tracing excursion widths of less than 10 dB; and 7. signed consent form plus verbal commitment to return 24 hours after the experiment for testing to assure complete recovery from TTS.

Instrumentation and Procedures

Exposure

Continuous broadband noise presented bilaterally at 90 dB SPL for two hours was used to represent noise exposure consistent with that commonly found in industrial environments.

Noise produced by a Coulbourn Instruments, model S81-02, noise generator was amplified (Coulbourn Instruments, S82-24 mixer-amplifier), attenuated (Hewlett-Packard, 350D attenuator set) and fed to Beyer DT48 earphones in the IAC booth.

Beyer earphones were chosen for their greater likelihood of producing constant SPL in the ear canal as opposed to a speaker in a sound-field. The Beyer earphone has a wide frequency response and is comfortable for long periods of wear.

The broadband noise spectrum used to produce TTS is included in figure 3 and was measured by coupling the earphone to a flat-plate attached to a standard 6 cc cavity as specified in a later section. A 1" B&K microphone, microphone amplifier and tape recorder

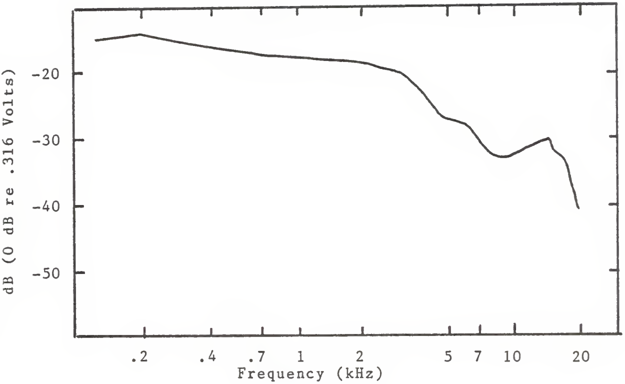


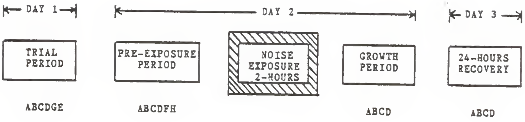
Figure 3. Spectrum of the TTS producing noise from the Beyer DT 48 earphone.

(AIWA 3800) were used to store the noise sample. This signal was later delivered to a PDP 11/23 computer and Princeton Applied Research (model 4512) FFT real time spectrum analyzer for spectral analysis and plotting.

Behavioral Hearing Threshold Measurements

Subjects were trained during the trial period to perform standard Bekesy tracking procedure for hearing threshold determination. Stable tracings for up to one minute with excursions of less than 10 dB were required before continuing. Thresholds were established for pulsed tones (250 msec with a 50% duty cycle) at discrete frequencies of 0.5, 1.0, 2.0, 3.0, 4.0, and 6.0 kHz. Stimuli were generated by a Hewlett-Packard 3311A function generator routed through a Coulbourn Instruments rise/fall gate and programmable attenuator and delivered to an earphone (Telephonics TDH 39, MX-41/AR). Digital logic circuitry was used with the programmable attenuator and an X-Y recorder to trace threshold.

Behavioral hearing thresholds in the right ear were established for all subjects during test intervals occurring prior to, immediately following, and finally, 24 hours after termination of the two hour noise exposure (figure 4). Sampling order for behavioral thresholds during each test interval was 6.0, 4.0, 3.0, 2.0, 1.0 and



A. Pure tone delivery system calibration for behavioral and acoustic reflex testing.

B. Broadband noise calibration for acoustic reflex testing and noise exposure.

C. Otoadmittance meter performance check (Exhaustive calibration was performed each week during data collection).

D. Behavioral threshold determination at 6.0, 4.0, 3.0, 2.0, 1.0 & 0.5 kHz.

E. Acoustic reflex decay determination during 7 1/2 minute exposure to broadband noise at 10 SL (re AR threshold for broadband noise). Baseline admittance drift determination for 7 1/2 minutes in the absence of elicitor stimulation.

F. Acoustic reflex threshold determination for 0.5, 1.0, 2.0, 3.0 & 4.0 kHz pure tone elicitors and broadband noise.

G. Acoustic reflex threshold determination for 1.0 kHz and broadband noise elicitors.

H. Two measures of acoustic reflex contraction strength at each level from threshold to 116 dB SPL (pure tones) and 106 dB SPL (broadband noise).

Figure 4. Time line representing test intervals with the measurements and order of sampling conducted during each interval. Letters below test periods refer to comments below the time line.

0.5 kHz. Tests conducted after exposure were obtained within 15 minutes following noise cessation.

TTS was calculated for each frequency by subtracting the pre-exposure behavioral threshold from thresholds measured during the two subsequent test intervals. Pre-exposure threshold was considered to be the value obtained during the initial test interval.

Acoustic Reflex Measurements

AR parameter measurements were obtained during trial and pre-exposure test intervals. Tympanometric data were collected to verify point-of-maximum-compliance and the external ear canal's physical volume before and after these test intervals. Susceptance (B) and conductance (G) data in millimhos were collected at +200 mm water and ambient pressure because changes in physical volume between the probe assembly and tympanic membrane have been reported to influence the magnitude of the AR (Wilson, Steckler and Jones, 1978). Probe placement was adjusted to assure constant physical volume within each subject. During the trial and pre-exposure test intervals, samples of reflex activity followed behavioral threshold measurements (figure 4).

The signals used to elicit the AR were generated by a Hewlett-Packard 3311A function generator routed through a

Coulbourn Instruments rise/fall gate, mixer/amplifier and a Hewlett-Packard 350D attenuator, and delivered to the same earphone used for behavioral thresholds. Stimuli were 1000 msec duration tones with a rise/fall of 20 msec. Contralateral AR activity was monitored with a Grason Stadler 1720 otoadmittance meter.

When the stimulus was presented, the voltage output of the otoadmittance meter (susceptance and conductance) was fed to a Data Translation, model DT2801A, analog-to-digital (A/D) converter. This information was digitized and stored in an IBM personal computer for later use. The A/D converter was set to sample 600 msec of baseline voltage before the reflex elicitor was presented and for a final 900 msec after the elicitor ended. Thus, each epoch had a 2500 msec duration. Upon visual inspection of the AR, the decision was made to either repeat the stimulus or proceed to the next signal. The judgment to repeat was based on the presence of any artifact that rendered the AR uninterpretable.

The signal was first introduced at a level where reflex activity was not expected and then increased in 2 dB steps until the AR was observed. If the AR was elicited during initial presentation, the stimulus level was adjusted until no AR activity was observed and then increased in 2 dB steps until AR response. The next signal following AR response was attenuated 4 dB and presented 3 successive times at that level. The signal level then was increased in 2 dB steps and the elicitor presented three times per level until a threshold response was determined.

To avoid interpretation problems in visual determination of AR threshold, the IBM computer was programmed to analyze variability in static baseline admittance activity. It established an AR response by comparing the baseline noise floor prior to stimulus delivery to the admittance during reflex contraction. When mean admittance during reflex activity exceeded the baseline value by more than two standard deviations, an AR response was recorded. Both susceptance (B) and conductance (G) recordings were examined and deviation by the prescribed amount in either was recorded as a response. The lowest intensity required to elicit a response at each frequency was considered threshold. Two responses out of the 3 presentations at one intensity level were required for threshold determination.

After AR threshold was established, an intensity function for each frequency was completed. The signal level was increased in 2 dB steps, two presentations at each level, until 12 dB SL. After this sensation level the intensity series continued in 2 dB steps, but with only one presentation at each level up to a maximum of 116 dB SPL (106 dB SPL for broadband noise).

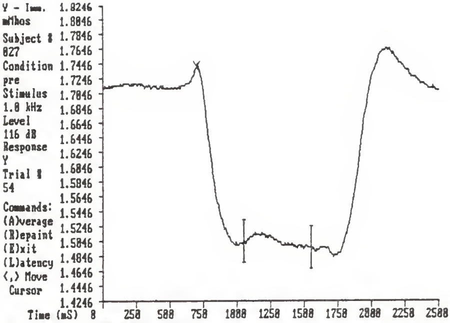
The magnitude of each acoustic reflex was computed in terms of acoustic admittance (Y). In this analysis, susceptance (B) and conductance (G) components were converted separately to static (B) and (G) by correcting for tympanometric data collected prior to AR testing. The acoustic admittance waveform was then calculated by the following formula:

$$Y = \sqrt{(G)^2 + (B)^2}$$

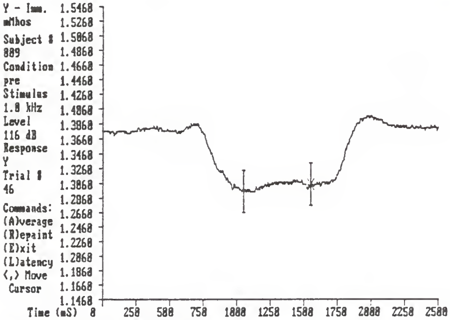
The difference in this waveform between average responses before and during the AR (in mmhos) served as the magnitude measure for each AR response. The average reflex activity of all samples represented the AR magnitude for each sensation level. Figure 5 shows the AR for two subjects. The elicitor in both cases is a 1.0 kHz pure tone presented at 116 dB SPL. The AR can be seen clearly as deviation in baseline static admittance activity. Variation between subjects in the magnitude of the AR response to the same elicitor also can be seen.

The computer was programmed to calculate onset latency by identifying the point at which the slope of contraction began for each response. In figure 5 this point is marked with an "X" in the top tracing. A measure of latency was recorded for each AR response.

Reflex decay was measured as the percent of change in AR magnitude (Y) as a function of time with reference to stimulus onset, a procedure outlined by Wilson, Steckler



```
Y - Imm.
mMhos
Subject #
027
Condition
pre
Stimulus
1.0 kHz
Level
116 dB
Response
Y
Trial #
54
Commands:
(A)verage
(R)epaint
(E)xit
(L)atency
(,) Move
 Cursor
Cursor at (x,y)=(701.4020,1.741549).
Latency point is at 86.40283 mSec.
```



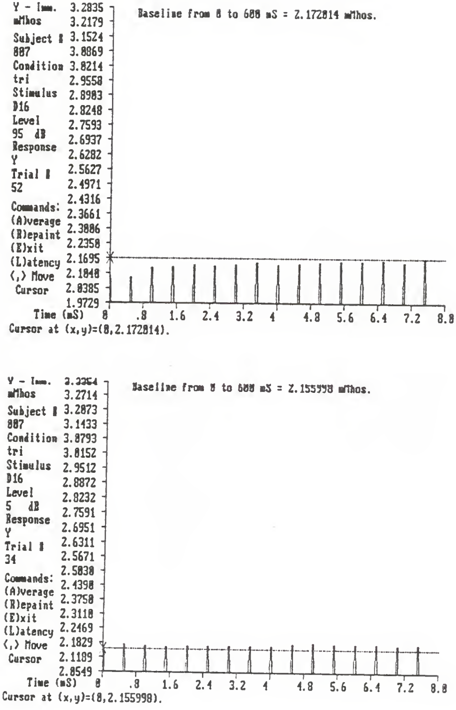
```
Y - Imm.
mMhos
Subject #
009
Condition
pre
Stimulus
1.0 kHz
Level
116 dB
Response
Y
Trial #
46
Commands:
(A)verage
(R)epaint
(E)xit
(L)atency
(,) Move
 Cursor
Cursor at (x,y)=(1553.106,1.304683).
Average from (1052.104,1.295305) to (1553.106,1.304683) is
        1.304042 mMho over 501.002 mS with S = 4.36732E-03 mMho.
```

Figure 5. Computer printout of the acoustic reflex elicited by 1.0 kHz at 116 dB SPL for two subjects.

and Jones, 1978. AR decay was monitored for 7 1/2 minutes during which the reflex was sustained with a broadband noise at 10 dB sensation level (re AR threshold). A 2500 msec segment of admittance activity was sampled by the computer every 15 seconds throughout the time periods. To assess baseline admittance stability over time, another 7 1/2 minute admittance sample without an acoustic signal was stored. Figure 6 shows the graphs of the decay (upper figure) and baseline drift (lower figure) series. Voltages were sampled at 15 second intervals and are presented as bars. Baseline admittance activity prior to elicitor presentation is shown as a horizontal dotted line. Voltages sampled at 15 second intervals reflect initial activation and decay of the AR. The bottom graph indicates variability in ongoing static admittance. Voltage differences across time depict fluctuations in baseline admittance activity. Again, the horizontal dotted line shows activity just prior to the 7 1/2 minute sample.

Calibration

The TDH-39 earphone used in this experiment was calibrated according to ANSI S3.7-1973 using a standard 1" Bruel and Kjaer condenser microphone coupled to a



```
Y - Imm.
mMhos
Subject #
007
Condition
tri
Stimulus
D16
Level
95  dB
Response
Y
Trial #
52
Commands:
(A)verage
(R)epaint
(E)xit
(L)atency
(,) Move
 Cursor
Baseline from 0 to 600 mS = 2.172814 mMhos.
Cursor at (x,y)=(0,2.172814).
```

```
Y - Imm.
mMhos
Subject #
007
Condition
tri
Stimulus
D16
Level
5  dB
Response
Y
Trial #
34
Commands:
(A)verage
(R)epaint
(E)xit
(L)atency
(,) Move
 Cursor
Baseline from 0 to 600 mS = 2.155998 mMhos.
Cursor at (x,y)=(0,2.155998).
```

Figure 6. Computer printout of acoustic reflex decay (top graph) and admittance baseline drift (bottom graph) for 7 1/2 minutes.

6cc cavity (type 4152). Microphone calibration was completed with a Bruel and Kjaer pistonphone (Model 4220).

Because of their non-standard cushions, the Beyer earphones could not be calibrated in the usual manner with a 6cc coupler. A flat-plate coupler was designed (figure 7) according to specifications published by Michael and Bienvenue (1976b). Calibration difficulty was encountered due to the airtight seal between the Beyer earphone and the flat-plate coupler which was positioned on top of a NBS 9-A coupler. A vent made from a small piece of catheter tubing (1/2 mm ID) was inserted between the Beyer earphone cushion and the flat-plate coupler to overcome this problem. Calibration was completed as recommended by Michael and Bienvenue (1976b).

Basic calibration procedures on the Grason-Stadler otoadmittance meter were performed per the instruction manual (User's Manual, Otoadmittance Meter, 1972). The output voltage was calibrated with a microsyringe such that 100 mV = 1 mmho at 220 Hz.

Data Analysis

Statistical analysis was carried out by an IBM 3081D computer using Statistical Analysis System (SAS) software. Standard descriptive statistics were obtained for all measures of AR parameters and behavioral threshold sensitivity. Correlational analysis was carried out between shifts in hearing threshold and each of the dynamic characteristics of the AR. This correlational analysis supplied scattergrams, coefficient of correlation, standard error (SE) of estimate and the slope and intercept for the linear regression equation. Multiple regression modeling also was conducted in an attempt to improve the predictive ability of AR measures.

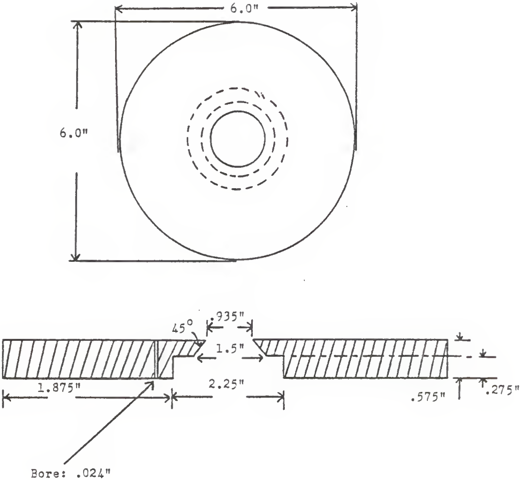


Figure 7. Flat plate used with the NBS 9-A coupler for the calibration of the Beyer earphone.

CHAPTER III
RESULTS

Behavioral Threshold Shift

Subjects exposed to 90 dB SPL broadband noise demonstrated statistically significant temporary threshold shift (TTS) at all test frequencies (paired-difference t test, alpha=.001) except 0.5 kHz. TTS was calculated by subtracting the pre-exposure behavioral threshold from the threshold measurement obtained after a two-hour noise exposure.

The mean threshold values at 0.5 through 4.0 kHz for all subjects during pre-exposure testing were within 2 dB of ANSI (1969) normal hearing values. Threshold values at 6.0 kHz were 5 dB higher than ANSI (1969). No significant differences (paired-difference t test) were noted between thresholds obtained during trial, pre-exposure, and recovery test intervals. Mean recovery thresholds were within 1 dB of pre-exposure thresholds at all frequencies indicating complete recovery 24 hours after exposure. Table 1 presents these results.

Table 1. Mean behavioral thresholds (dB SPL) and standard devitions for pre-exposure and recovery test intervals (N=37). ANSI (1969) standards are also presented.

	Pre-exposure		Recovery		
Frequency (kHz)	Threshold dB SPL (X)	Standard Deviation (SD)	Threshold dB SPL (X)	Standard Deviation (SD)	ANSI Standards dB SPL
0.5	10.86	4.2	10.84	4.7	11.5
1.0	6.65	5.2	6.05	5.2	7.0
2.0	9.70	6.3	9.84	6.4	9.0
3.0	8.16	5.0	8.81	4.9	10.0
4.0	9.38	6.4	10.40	6.5	9.5
6.0	21.89	7.8	22.30	6.8	15.5

Figure 8 shows the means and standard deviations of TTS by frequency. As expected, the greatest amount of TTS averaged across all subjects occurred at 4.0 kHz and was 19.3 dB. Typically some subjects showed extensive TTS while others had little and this spread is illustrated in figure 9.

Although the average TTS was greatest at 4.0 kHz, individual subjects varied in the frequency most sensitive to TTS. Some demonstrated greatest TTS at 3.0 kHz, others at 4.0 kHz, and still others at 6.0 kHz. By describing the deleterious effects of broadband noise exposure as TTS only at an individual frequency, some subjects' sensitivity to noise would be understated. Therefore, to compare TTS to various properties of the ear, each individual's TTSs at all frequencies were added and expressed as Total TTS. Likewise, it is also reasonable to assume that, due to these differences in maximum TTS frequency, individual variations in properties of the ear may cause one person's high frequency hearing to be at risk while the risk to another may be in the lower frequencies. This delineation would be extremely important if definition of risk encompassed a three-frequency average (e.g. 1 + 2 + 3 kHz or 3 + 4 + 6 kHz) for noise induced loss compensation cases. Table 2 presents a linear correlation matrix showing the relationship between different TTS measures. Table 3

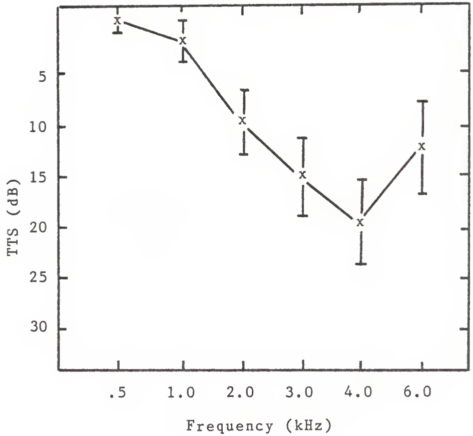


Figure 8. Average TTS across 37 subjects (bar = one standard deviation).

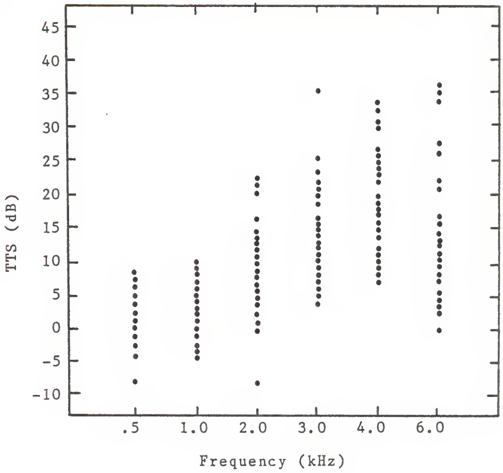


Figure 9. Individual TTS by frequency (N = 37).

Table 2. Correlation matrix for different measures of temporary threshold shift.

TTS (kHz)	Total TTS	6.0	4.0	3.0	2.0	1.0
Total TTS	1.0	*.59	*.73	*.73	*.58	*.42
6.0		1.0	.29	.16	.11	.00
4.0			1.0	*.51	.20	.19
3.0				1.0	*.44	.17
2.0					1.0	.26
1.0						1.0

* alpha = .01

presents descriptive statistics for different frequencies and frequency combinations of TTS. Regardless of the method used to define effects of noise on behavioral hearing sensitivity, the variability in TTS across subjects is striking. It should be noted that there is an associated variance with each of the individual components that comprise measures of TTS. Since TTS combinations are the sum of these components and variance tends to be additive, there is the potential for inflated variability in Total TTS or other TTS combinations. The presence of this excessive variation could obscure correlations between properties of the ear and expressions of TTS. However, since risk is typically defined as an average hearing loss across frequency, it seems appropriate to analyze TTS in this manner. For this study, Total TTS, TTS 1 + 2 + 3 kHz, TTS 2 + 3 + 4 kHz, and TTS 3 + 4 + 6 kHz were used in all correlational analyses.

Acoustic Reflex Properties and TTS

Acoustic Reflex Threshold

Acoustic reflex (AR) response was defined as a shift of at least two standard deviations in mean static acoustic admittance in the presence of an activating signal. The lowest signal level for each elicitor needed

Table 3. Descriptive statistics for different frequency and frequency combinations of temporary threshold shift.

Expression of TTS (kHz)	Minimum Score (dB)	Maximum Score (dB)	Mean (dB)	Standard Deviation (dB)
Total TTS	12	107	59.08	22.19
3 + 4 + 6	17	89	46.95	16.46
2 + 3 + 4	9	80	42.68	15.70
1 + 2 + 3	6	56	26.32	12.87
4 + 6	6	54	32.08	12.44
2 + 3	3	50	23.35	11.25
6	0	35	12.76	8.29
4	6	33	19.32	7.19
3	3	35	14.86	6.88
2	-8	23	8.49	6.36
1	-5	10	2.97	4.00

to create this criterion shift was identified as AR threshold. This response has also been referred to as "criterion magnitude" by other researchers (Block and Wiley, 1979).

Table 4 presents descriptive statistics for AR thresholds obtained during the pre-exposure test interval. Again, there was considerable variability across subjects. These data are in agreement with threshold values reported by other investigators (Chun and Raffin, 1979; Gerhardt and Hepler, 1983).

Simple linear regression was used to answer the research question, "can AR thresholds be used to explain variation in TTS?" No significant relationship was found between any definition of TTS and AR threshold for the elicitors used in this study. The 4.0 kHz pure tone failed to elicit a recordable AR in some subjects. The use of maximum intensity (116 dB SPL) as the threshold for averaging in these subjects could bias correlational analysis. Since AR thresholds and TTS failed to correlate for other elicitors, it seems unlikely that any correlation would exist for the 4.0 kHz elicitor regardless of the method used to deal with the unknown thresholds.

Table 4. Descriptive statistics for acoustic reflex pre-exposure thresholds.

Elicitor	Minimum Threshold (dB)	Maximum Threshold (dB)	Mean (dB)	Standard Deviation (dB)
0.5 kHz	74	114	99.57	8.63
1.0 kHz	85	108	96.54	5.93
2.0 kHz	88	114	98.94	6.38
3.0 kHz	86	116	98.58	7.71
4.0 kHz	90	116*	103.06	8.49
Broadband Noise	60	100	81.32	10.90

* This level produced no AR in some subjects. Maximum intensity was used for averaging when there was no recordable response.

Acoustic Reflex Magnitude

Estimates of AR magnitudes were obtained by creating intensities up to 116 dB SPL for pure tone elicitors and 106 dB SPL for the broadband noise elicitor. The starting point for the intensity series was AR threshold, with magnitude recorded in 2 dB steps.

Individual variability in AR magnitude was quantified in several ways. This data was used to discover an optimum definition of magnitude that would effectively characterize differences across subjects. One definition considered was the AR intensity function slope (mmho change per dB increase in elicitor level). Another was individual peak magnitude (in mmhos), regardless of sensation level, for each elicitor. Average AR magnitude was calculated for responses obtained at the 6, 8, 10, and 12 dB sensation level for each elicitor an expression similar to that used by Gerhardt and Hepler (1983).

Before exposure, the average AR magnitude elicited by broadband noise accounted for the highest percentage of variation in TTS. Simple linear correlational analysis indicates that 16% ($r = -.40$) of the variability in total TTS could be accounted for by this expression of magnitude (significant at the alpha = .01 level). Table 5 presents the relationship between other measures of TTS and average AR magnitude created by the broadband noise elicitor.

Table 5. Linear regression results for temporary threshold shift and pre-exposure average acoustic reflex magnitude elicited by broadband noise.

	Average Reflex Magnitude		
Expression of TTS (kHz)	Correlation Coefficient (r)	Coefficient of Determination (r^2)	Attained Significance Level (alpha)
Total TTS	-.40	.16	.01
3 + 4 + 6	-.40	.16	.01
2 + 3 + 4	-.36	.13	.03
1 + 2 + 3	-.31	.10	.06

AR magnitude elicited by broadband noise was found to be consistently related to most TTS measures. This was true for individual magnitude values for a single sensation level and for averaged magnitude data. All correlations were in a negative direction, indicating the more vigorous the acoustic reflex, the smaller the behavioral threshold shift caused by the two-hour broadband noise exposure. These results agree with results published by Gerhardt and Hepler (1983).

The AR magnitude/intensity function slope and peak AR magnitude elicited by broadband noise, as well as magnitude values elicited by pure tones, were not consistently related to TTS caused by broadband noise. Isolated correlations proved significant to one TTS value or another. However, considering the number of correlations executed, the element of chance could account for these significant relationships.

Although measures of AR magnitude elicited by pure tones proved to be less predictive of TTS, a trend existed in all linear regression analyses. As in correlations between TTS and AR magnitude elicited by broadband noise, all correlation coefficients were in a negative direction. This trend supports the conclusion that the AR influences the level of TTS.

Two additional findings concerning AR magnitude were noteworthy. AR magnitude was consistent within subjects across elicitors. In other words, the subject with a very strong AR created by one elicitor would also exhibit a vigorous response to a different elicitor. For example, AR magnitude created by the 0.5 kHz elicitor at 10 dB sensation level (SL) was significantly related to the magnitude elicited by 1.0 kHz at 10 dB SL (r=.82, alpha=.0001). It was also related to the 2.0 kHz, 10 dB SL response (r=.68, alpha=.0002), and so forth. Likewise, AR magnitude created by the broadband noise elicitor was related to the magnitude elicited by pure tones. Although significant, this correlation was not as strong (0.5 kHz - r=.42; 2.0 kHz - r=.59, etc.). The correlation between one magnitude and another was weakest for the 4.0 kHz elicitor. This level also was the most variable in terms of eliciting AR threshold and magnitude. Average threshold for the 4.0 kHz elicitor was 103 dB SPL with 15 subjects exhibiting thresholds greater than 112 dB SPL. Other investigators have noted variability of the 4.0 kHz elicitor, most recently Chermak, Dengerink and Dengerink (1983).

The second finding concerning AR magnitude was its relationship to static middle-ear admittance. For pure tone elicitors, many values of individual AR magnitude and all measures of slope, average and peak magnitude were significantly correlated to static middle-ear admittance. The higher the static admittance, the stronger the AR magnitude elicited by pure tones (average r=.54 and alpha=.01). This same relationship did not exist for broadband noise. In addition, there was no significant correlation for static middle-ear admittance and any measure of TTS. This last finding was expected since magnitude elicited by broadband noise was correlated to TTS while static middle-ear admittance was not related to broadband noise magnitudes.

There was considerable variation in the external ear canal volume among the 37 subjects. Analysis indicated no significant correlation between measures of AR magnitude and ear canal volume.

Acoustic Reflex Latency

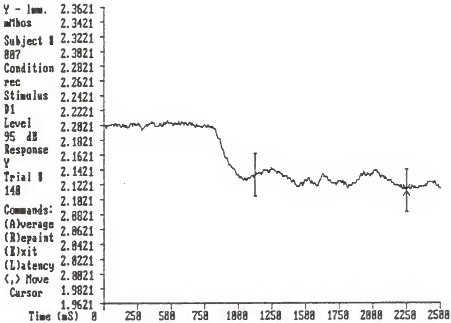
In contrast to AR magnitude data, values of latency were confounded with artifact. Even with computer assistance it was difficult to consistently identify valid latency measures. Latency decreased with increased elicitor levels, a trend easily seen when the latency/intensity function was examined across subjects. However, when this function was examined in discrete steps, considerable variability in individual data points was often noted.

A computer program was devised to identify latency and eliminate experimenter bias in latency determination.

In all designated AR responses, the computer identified the rapid change in voltage caused by AR. Latency was labeled as the point where this slope intersected mean baseline admittance. Because of the latency variability, individual values were averaged across intensity for each elicitor. This allowed an initial approximation of individual differences in AR latency. Linear regression was used to model latency and various measures of TTS. No significant linear relationships were found.

Acoustic Reflex Decay

Broadband noise was chosen as the AR decay elicitor because it was the fatiguing stimulus used for this study. Baseline admittance without elicitor stimulation was monitored for each subject. The intent was to show stability in static admittance, or at least provide a correction factor upon which accurate decay information could be based. This baseline drift was sampled for 7 1/2 minutes to match the time course of the decay series. Unfortunately, static admittance proved unstable over time. The bottom graph in figure 10 illustrates this fluctuation. The graph shows average variability in static admittance, but many subjects exhibited marked variation. Due to this drift in static admittance, calculation of decay as a shift from, and recovery to,



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Y - Imm.
mMhos
Subject #
007
Condition
rec
Stimulus
D1
Level
95  dB
Response
Y
Trial #
140
Commands:
(A)verage
(R)epaint
(E)xit
(L)atency
(,) Move
 Cursor
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```
Cursor at (x,y)=(2254.509,2.114077).
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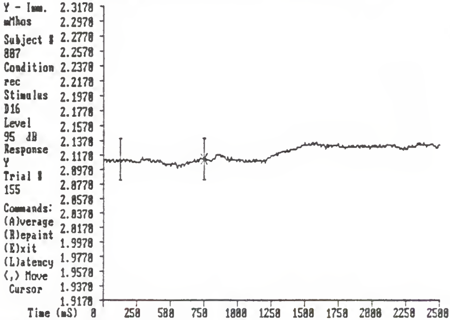

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Y - Imm.
mMhos
Subject #
007
Condition
rec
Stimulus
D16
Level
95  dB
Response
Y
Trial #
155
Commands:
(A)verage
(R)epaint
(E)xit
(L)atency
(,) Move
 Cursor
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Average from (125.2505,2.112410) to (751.5031,2.112410) is
        2.100223 mMho over 626.2526 mS with S = 3.762990E-03 mMho.
```

Figure 10. Computer printout of AR onset and off-set which occurred during a 7 1/2 minute decay series.

original baseline admittance, proved impossible. In most cases, baseline drift exceeded the admittance change caused by fatigue of the AR. Because of these difficulties, a second method of decay computation was devised.

The first 2500 msecs of the decay series, as in all samples of AR activity, allowed for a 600 msec segment of baseline admittance prior to presentation of the elicitor. From this first decay series sample (the top graph in figure 10 is an example), an onset response could be determined. The response magnitude was calculated and served as the reference (100%) point for AR decay. The bottom graph in figure 10 shows an example of the last sample in a decay series. The elicitor was programmed to terminate mid-way through this final 2500 msec period. Magnitude could then be determined at signal off-set and calculated with reference to the baseline established after signal termination. The off-set magnitude (M2) was expressed as a percentage decline from onset magnitude (M1) by the following formula:

$$\text{Decay} = 1 - (M2/M1) \times 100$$

This percentage served as the measure of decay for each subject. Average decay for the 37 subjects was 83.4% with a range of 31% to 100%. Table 6 presents linear regression results for TTS and AR decay elicited by

Table 6. Linear regression results for temporary threshold shift and pre-exposure acoustic reflex decay elicited by broadband noise.

	Average Reflex Decay		
Expression of TTS (kHz)	Correlation Coefficient (r)	Coefficient of Determination (r^2)	Attained Significance Level (alpha)
Total TTS	-.33	.11	.05
3 + 4 + 6	-.28	.08	.09
2 + 3 + 4	-.18	.03	.29
1 + 2 + 3	-.29	.08	.09

broadband noise. Decay was significantly related (alpha=.05) to total TTS and accounted for approximately 11% of its variability (r=.33).

Onset magnitude (M1) was analyzed to test the hypothesis that subjects with large onset magnitudes would suffer more AR decay. Regression analysis indicated no significant relationship between initial magnitude and decay (r=.0006).

Behavioral Thresholds and TTS

Linear regression analysis indicated a significant negative relation between pre-exposure behavior thresholds and TTS at 1.0, 2.0, 3.0, 4.0, and 6.0 kHz. As behavioral threshold increased within the normal range, TTS decreased. In addition, slopes of the regression lines tend to be steeper at higher frequencies. This is probably due to the increase in TTS at higher frequencies. These results are consistent with Ward's 1965 findings in subjects with sensori-neural hearing loss and, most recently, with Davis and Ahroon (1972) in subjects with normal hearing. Figures 11 through 16 illustrate these relationships for each frequency. Correlation coefficients, attained significance levels, and the predictive linear regression formulas are also provided.

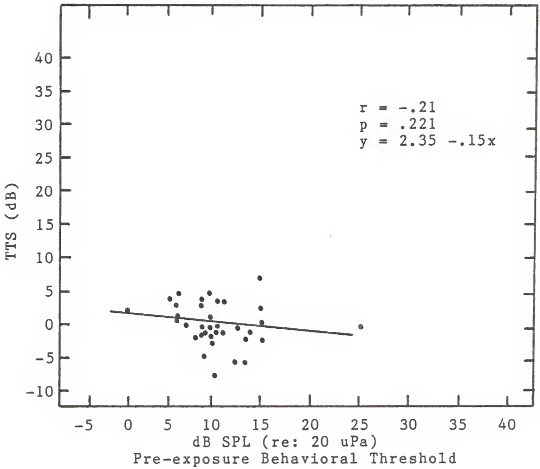

Figure 11. Individual TTS by pre-exposure behavioral threshold at 0.5 kHz.

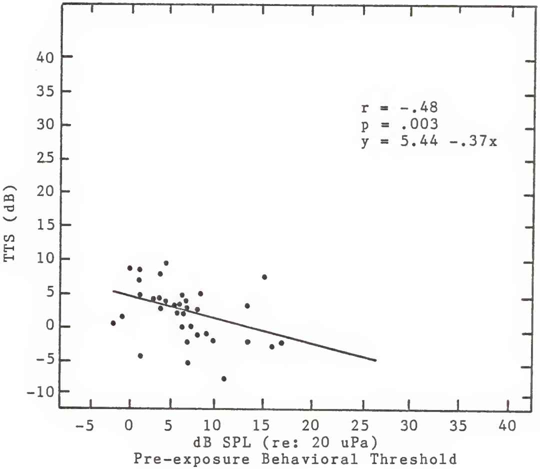

Figure 12. Individual TTS by pre-exposure behavioral threshold at 1.0 kHz.

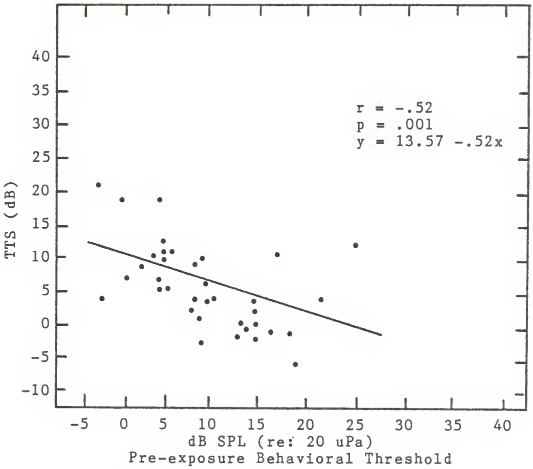

Figure 13. Individual TTS by pre-exposure behavioral threshold at 2.0 kHz.

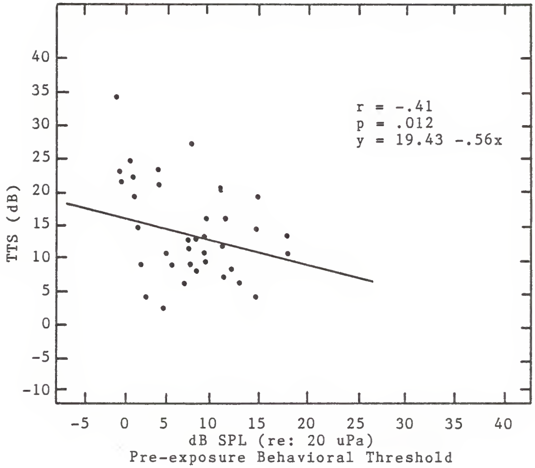

Figure 14. Individual TTS by pre-exposure behavioral threshold at 3.0 kHz.

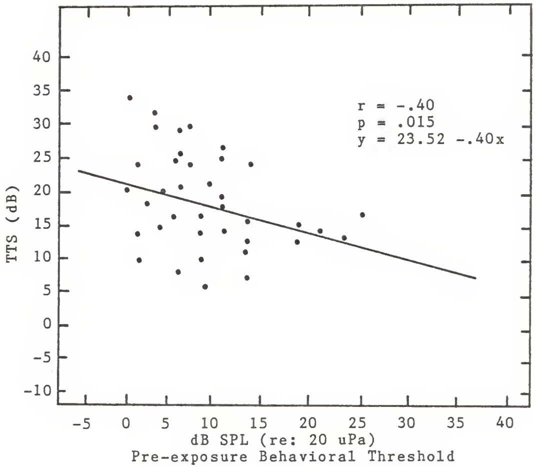

Figure 15. Individual TTS by pre-exposure behavioral threshold at 4.0 kHz.

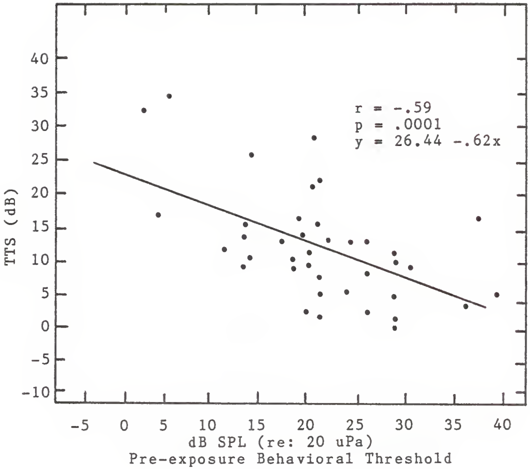

Figure 16. Individual TTS by pre-exposure behavioral threshold at 6.0 kHz.

Pre-exposure thresholds were not significantly related to total TTS in a linear regression model. Behavioral threshold at 3.0 kHz related to the 3 + 4 + 6 kHz expression of TTS (r=-.44), while 2.0 kHz related to the 2 + 3 + 4 kHz expression (r=-.37) and 1 + 2 + 3 kHz measure of TTS (r=-.37). Other correlations were not significant. These data suggest the predictive ability of pre-exposure behavioral thresholds is obscured when TTS is collapsed across frequency.

Ear Canal Volume and TTS

The majority of subjects in this study suffered the greatest TTS at 4.0 kHz. However, some subjects demonstrated maximum TTS at 3.0 kHz and others at 6.0 kHz. This is a common clinical finding in the audiometric configurations of patients who suffer permanent noise-induced hearing loss. The reason for this difference in the frequency most susceptible to noise damage has never been clearly established. The study results indicated a relationship between ear canal volume and the frequency of maximum TTS. Figure 17 presents ear canal volume estimates for subjects with maximum TTS occurring at 3.0, 4.0 and 6.0 kHz. Analysis of variance performed on these data indicated a significant difference between groups (see table 7). The Duncan post-hoc test

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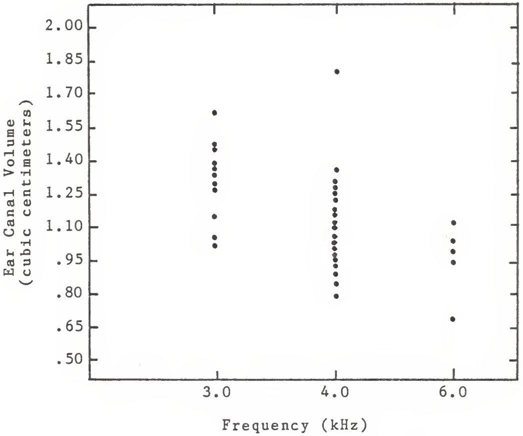

Figure 17. Estimate of ear canal volume by frequency of maximum TTS. Volume is estimated from admittance recorded while the middle-ear system is stiffened with -200 mm H_2O equivalent air pressure (1 cc=1 mmho).

Table 7. ANOVA table comparing ear canal volume and frequency of maximum temporary threshold shift.

Source	df	SS	MS	F	PR>F
Volume	2	.353	.176	4.13	.0247
Error	34	1.450	.043		
Total	36	1.803			

was applied and significant differences in ear canal volume were found between groups of subjects with maximum TTS at 3.0 kHz and those with maximum TTS at 6.0 kHz. Subjects with maximum TTS at 6.0 kHz had smaller ear canal volumes than subjects with maximum TTS at 3.0 kHz.

Non-Auditory Information and TTS

During the trial test interval various non-auditory information was obtained on each subject. These variables were analyzed for their influence on TTS development. Linear regression analyses between age and measures of TTS were not significant. Parceling was conducted by race (white/black) and eye color (blue/green/brown). T-tests comparing the two races on measures of TTS indicated no significant differences and analysis of variance comparing eye colors on measures of TTS also were not significant.

At 3.0 kHz, males differed significantly (alpha=.05) from females in the slope of the pre-exposure AR magnitude intensity function. Two sample T-tests for independent samples also indicated significant differences (alpha=.05) between males and females in peak AR magnitude at 3 kHz and individual magnitude values at 4, 6, 8, 10, 12, 14, and 16 SL. Females consistently showed a more vigorous AR at this frequency. No significant differences were noted for the other pure tone or broadband noise elicitors. A significant difference (alpha=.01) was noted between males and females in volume of the ear canal. Male ear canal volume was larger than female and males exhibited significantly (alpha=.05) more TTS at 3.0 kHz and at 1 + 2 + 3 kHz than females. Males and females did not differ significantly in other expressions of TTS. Additional research is needed to analyze sex as a possible covariate in relating ear canal volume to frequency of maximum TTS. A definitive answer to this possible relationship could not be obtained from these data.

Multiple Regression Modeling

During correlational analyses, middle-ear muscle parameters and other properties of the ear were found to significantly relate to measures of TTS. However, the correlation coefficients (r) were small. Multiple regression analysis was used to improve the predictive relation between properties of the ear and each expression of TTS.

A stepwise modeling procedure designed for maximum improvement in the coefficient of determination (r^2) was selected. The maximum r^2 improvement technique first determined the best one-variable model and then the remaining variables were combined with the first variable until the best two-variable model was found. The process continued until all variables were added to each model (SAS User's Guide, 1979).

This technique differs from other modeling procedures in that it does not settle on one model, but rather searches for the best one-variable model, two-variable model, etc. A standard SAS program presents a complete analysis on each model and includes: coefficient of determination; degrees of freedom; sums of squares and mean squares; F statistic and overall significance level; intercept and slopes for individual variables; and type II sums of squares for each variable in the model with respective F statistics and significance levels.

Table 8 presents the linear regression predictive models which yielded the highest coefficient of determination for combinations of TTS. For three measures of TTS the data suggested an expression of AR magnitude elicited by broadband noise as the best predictor. When TTS was averaged for high frequencies, a pre-exposure behavioral threshold at 3.0 kHz was the best predictor.

Two methods were used to select a multiple regression model most appropriate for predicting each expression of TTS. A conservative approach was taken using a comparison of the sums of squares for error in complete and reduced models. In this procedure, a multiple regression model was selected as "best" only if the addition of new variables significantly (alpha=.05) reduced the sum of

Table 8. Linear regression predictive models that yield the highest coefficient of determinations for temporary threshold shift.

Expression of TTS	Best Variable	Linear Regression Model	Coefficient of Determination	Attained Significance Level
Total TTS	Average AR Magnitude to Broadband Noise	Y= 46 - 89(X)	.16	.0192
3 + 4 + 6	Pre-exposure Behavioral Threshold 3 kHz	Y= 59 - 1.4(X)	.20	.0075
2 + 3 + 4	Average AR Magnitude to Broadband Noise	Y= 35 - 54(X)	.12	.0458
1 + 2 + 3	Initial AR Magnitude of Decay Series	Y= 22 - 75(X)	.12	.0377

squares for error in the predictive model. Table 9 shows the best multiple regression models for predicting TTS with this procedure. Additional variables did not significantly improve Total TTS prediction; however, the prediction of other expressions of TTS were significantly improved by adding other variables to the model.

Initial AR magnitude during the decay series proved to be the best TTS predictor at 1 + 2 + 3 kHz in a linear model. However, when additional variables were added, other properties of the ear were found to be more predictive. The three-variable model selected as "best" for this measure of TTS was compared to reduced models of these same variables.

The second method for selecting an appropriate multiple regression model to predict TTS was not as statistically demanding as the first one. In this procedure, the model was chosen that produced the highest coefficient of determination while maintaining an overall significance level (for the entire model) below alpha=.05. Table 10 presents these results. The overall significance level for a multiple regression model addresses only the probability that at least one slope in the equation is not zero. The probability that each individual slope is significantly different from zero may not be .05. In these models, most of the significance levels for individual slopes exceeded .05. Although not as

Table 9. Multiple regression predictive models that yield the highest coefficient of determinations for temporary threshold shift using complete/reduced model comparison technique.

Expression of TTS	Best Variable	Linear Regression Model	Coefficient of Determination	Attained Significance Level
Total TTS	There are no multiple regression models that produce significantly better results than the linear model.			
3 + 4 + 6	X1-Pre-exposure Behavioral Threshold 3 kHz X2-Average AR Magnitude to Broadband Noise	$Y = 49 - 1.2(X1)$ $- 52(X2)$	.29	.0044
2 + 3 + 4	X1-Average AR Magnitude to Broadband Noise X2-Average AR Latency to Broadband Noise	$Y = 20 - 66(X1)$ $+ .09(X2)$	.24	.0164
1 + 2 + 3	X1-Decay X2-Volume of Ear Canal X3-Pre-exposure Behavioral Threshold 2 kHz	$Y = -10 + .26(X1)$ $+ 18(X2)$ $- .69(X3)$	.30	.0101

Table 10. Multiple regression predictive models that yield the highest coefficient of determinations for temporary threshold shift using overall significance level for comparisons.

Expression of TTS	Best Variable	Linear Regression Model	Coefficient of Determination	Attained Significance Level
Total TTS	X1-Decay X2-Volume of Ear Canal X3-Pre-exposure Behavioral Threshold 2 kHz X4-Pre-exposure Behavioral Threshold 4 kHz X5-Average AR Magnitude to Broadband noise	Y= 7 + .51(X1) + 13(X2) - .78(X3) - .81(X4) - 63(X5)	.31	.0443
3 + 4 + 6	X1-Volume of Ear Canal X2-Middle-ear Admittance X3-Pre-exposure Behavioral Threshold .5 kHz X4-Pre-exposure Behavioral Threshold 1 kHz X5-Pre-exposure Behavioral Threshold 3 kHz X6-Pre-exposure Behavioral Threshold 6 kHz X7-Average AR Magnitude to Broadband Noise X8-AR Reflex Threshold to Broadband Noise	Y= 44 + 17(X1) - 28(X2) - 1(X3) + 54(X4) - 1(X5) - .57(X6) - 59(X7) + .21(X8)	.44	.0353

Table 10–continued.

Expression of TTS	Best Variable	Linear Regression Model	Coefficient of Determination	Attained Significance Level
2 + 3 + 4	X1–Volume of Ear Canal X2–Middle-ear Admittance X3–Pre-exposure Behavioral Threshold 2 kHz X4–Pre-exposure Behavioral Threshold 3 kHz X5–Average AR Magnitude to Broadband Noise X6–Average AR Latency to Broadband Noise	Y= 27 + .15(X1) – 25(X2) – .58(X3) – .48(X4) – 60(X5) + .07(X6)	.36	.0399
1 + 2 + 3	X1–Decay X2–Volume of Ear Canal X3–Pre-exposure Behavioral Threshold .5 kHz X4–Pre-exposure Behavioral Threshold 2 kHz X5–Average AR Magnitude to Broadband noise X6–Pre-exposure AR Reflex Threshold to Broadband Noise X7–Average AR Latency to Broadband Noise	Y= –9 + .23(X1) + 17(X2) + .71(X3) – .79(X4) – 22(X5) – .16(X6) + .04(X7)	.38	.0489

statistically defendable, in a preliminary study these models are useful in analyzing trends and offering support to conclusions based upon other analyses. Approximately 40% of the variation in TTS can be explained using this modeling procedure.

The relationships found between TTS and the properties of the ear in this study are consistent in all linear and multiple regression models. Measures of pre-exposure AR magnitude are all negatively related to expressions of TTS. In addition, AR magnitude provides the strongest prediction of TTS. These data support the hypothesis that the more vigorous the AR the less will be the deleterious effects caused by exposure to hazardous noise. Middle-ear admittance and, generally, pre-exposure behavioral thresholds were also negatively related to measures of TTS. AR decay and ear canal volume, on the other hand, were directly related to TTS. As AR decay and middle-ear volume increased, so did TTS.

Parceling of Subjects by TTS and AR Properties

To further explain the relation between properties of the AR and TTS, the 37 subjects were parceled into two groups for certain variables: 1) Total TTS, 2) TTS 1 + 2 + 3 kHz, 3) TTS 2 + 3 + 4 kHz, 4) TTS 3 + 4 + 6 kHz, 5) AR decay and, 6) average AR magnitude elicited by broadband noise. In analyzing each variable, the first group contained subjects with the highest seven values and the second group the lowest seven values (approximately 20%). The measures of TTS were analyzed for differences in properties of the AR, and the AR variables were tested for differences in TTS.

This parceling by extremes after data collection has obvious flaws in sampling methodology. Statistically, the samples are biased and the validity of individual T-comparisons can be questioned. But again, because of the explorative nature of this study, this information is valuable for supporting trends in the data. In summary, previous data analyses have indicated a low, but statistically significant predictive relation between TTS and properties of the AR. Magnitude has shown a negative relationship, while decay has shown a positive relationship. AR threshold and latency have provided the least information about TTS while the TTS measures have proven to be highly interrelated. The difference in the predictive power of one measure of TTS over another seems to be based upon frequency relationships between the expressions of TTS. Other measures of the ear (eg. behavioral thresholds, ear canal volume, and middle-ear admittance) have provided additional predictive information about TTS.

Another reasonable question would be: Do any of these properties of the ear contribute to the development of TTS? This study was not designed to directly answer this question; however, parceling the data in the manner described could lend insight into the designs for studies that directly investigate this question.

Subjects parceled on Total TTS were examined for differences in TTS at each behavioral test frequency. Figure 18 presents these results. TTS differences at all frequencies were striking. As expected, subjects with high Total TTS also showed consistently high TTS at each individual frequency.

Regardless of the expression of TTS, average AR magnitude elicited by broadband noise was consistently different between extremes. Also, for TTS 2 + 3 + 4 kHz, individual magnitudes elicited by pure tones (0.5 and 1.0 kHz) were statistically significant. Figure 19 presents idealized waveforms representing average AR magnitude elicited by broadband noise for subjects with the greatest and least Total TTS. Subjects with the greatest magnitude suffered the least TTS.

AR decay was significantly different between the extremes for subjects parceled by Total TTS and TTS 1 + 2 + 3 kHz. When TTS was analyzed by high frequency combinations, decay was no longer a significant variable. Figure 20 typifies decay differences between the two

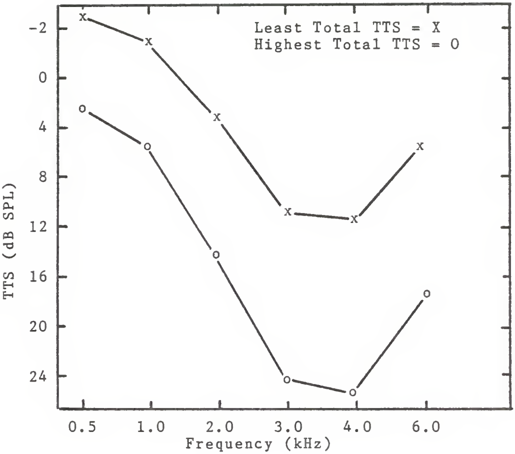

Figure 18. Average TTS by frequency of subjects with highest and lowest total TTS (N = 7 for each group).

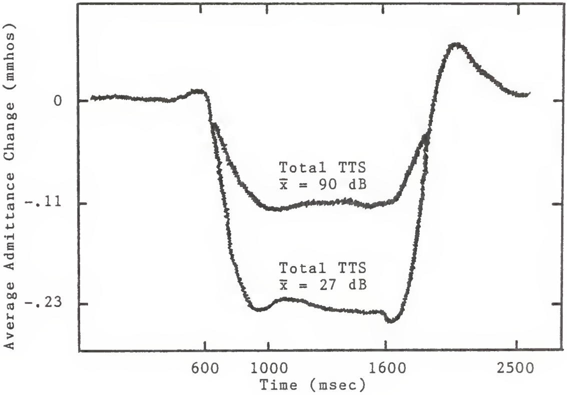

Figure 19. Idealized waveform representing the AR magnitudes elicited by broadband noise (6,8,10 & 12 SL) from the groups of subjects with the greatest and least TTS.

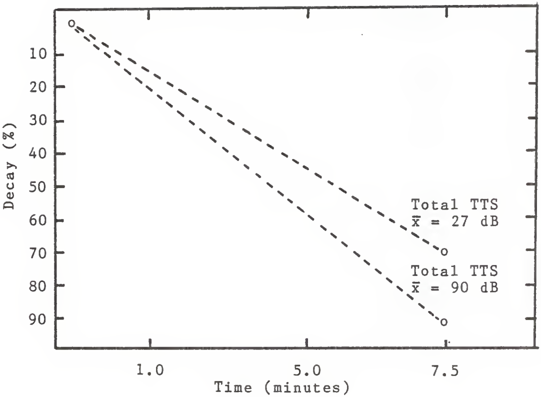

Figure 20. Average decay for AR magnitude elicited by broadband noise (10 SL) for subjects with highest and lowest total TTS (N = 7 for each group).

groups for Total TTS. Subjects with the greatest decay suffered the greatest TTS.

Differences in behavioral thresholds, ear canal volume and middle-ear admittance which proved predictive of TTS in regression analyses could not differentiate between groups divided by extreme values of TTS. Occasionally an individual T-comparison was significant, but there were no consistent trends in the data.

When subjects were divided by extreme values in average AR magnitude elicited by broadband noise and AR decay, the results supported the data found in parceling by TTS. Table 11 presents average TTS comparisons between groups with high and low values in these AR properties.

Groups divided by average AR magnitude elicited by broadband noise were also significantly different on most individual magnitude values, regardless of the elicitor. For example, subjects with the highest average AR magnitude elicited by broadband noise also had high AR magnitude when the elicitor was 0.5 or 1.0 kHz. This was true for all elicitors but 4.0 kHz. Magnitude of the AR caused by the 4.0 kHz elicitor did not seem to relate to other expressions of AR magnitude.

Table 11. Average TTS between low and high groups parceled on average AR magnitude elicited by broadband noise and AR decay.

Expression of TTS (kHz)	Average Magnitude Low ($\bar{X}$)	Average Magnitude High ($\bar{X}$)	Decay Low ($\bar{X}$)	Decay High ($\bar{X}$)
Total TTS	79.4	34.0	48.6	75.3
3 + 4 + 6	63.9	31.3	40.0	58.2
2 + 3 + 4	52.6	26.0	39.1	52.2
1 + 2 + 3	33.0	14.5	22.7	35.8

CHAPTER IV
DISCUSSION

Interpretation of Results

Behavioral Threshold Shift

Average TTS developed by the 2-hour exposure (figure 8) is consistent with the results of previous investigators (U.S. Environmental Protection Agency, 1971; Ward et al., 1976; Mills et al., 1981; Gerhardt and Hepler, 1983). Furthermore, these authors noted wide individual variability in TTS which was also found in the current study. Some subjects were especially susceptible to threshold shift while others were more resistant. As expected, this exposure produced a marked shift in hearing sensitivity from 2.0 kHz to 6.0 kHz with the maximum TTS occurring at 4.0 kHz. The "worst-case" TTS for these subjects was 7 to 10 dB higher than predicted by the U.S. Environmental Protection Agency Report (1971) and may have been a function of the Beyer earphone and calibration procedure. Recent research has shown that a signal delivered through the Beyer earphone is approximately 5 to 7 dB louder than the same signal from the TDH 39 earphone (Burnett, 1984). The maximum shifts in hearing sensitivity noted are more consistent with exposures of up to 95 dB SPL.

TTS was expressed as a total for all frequencies and as a total for specific frequency combinations. Because of TTS variability by frequency, expressions for total TTS were used. There was little consistency in TTS magnitude across frequency for each subject (table 2). Therefore, the relationship between TTS and various properties of the ear would vary dependent upon the frequency chosen to measure TTS. For example, if TTS at 6.0 kHz represented the deleterious effects of the 90 dB SPL broadband noise exposure, Subject Two would be at considerable risk (34 dB). However, if TTS at 2.0 kHz was used, Subject Two would have a very low risk (0 dB). According to the U.S. Environmental Protection Agency (1979), most definitions of noise damage entail averaging of hearing threshold shift across frequency (e.g. National Institute of Occupational Safety and Health, NIOSH, 1 + 2 + 3 kHz; American Medical Association, AMA, 1947, .5 + 1 + 2 + 4 kHz; American Academy of Opthalmology and Otolaryngology, AAOO, 1979, .5 + 1 + 2 + 3 kHz; and Oregon Formula, .5 + 1 + 2 + 4 + 6 kHz). There would be little demand for a susceptibility index that successfully predicts TTS at only one frequency. However, it would be useful to know if the averaging technique used to define noise-induced hearing loss affected the susceptibility ranking of an individual. Would a person identified as susceptible by AMA standards be judged equally at risk by another standard? TTS expressions analyzed were intended to provide information on this issue.

Subject variations were found in the frequency of maximum TTS. Some subjects showed a maximum change in hearing sensitivity at 3.0 kHz and others at 4.0 or 6.0 kHz. This difference was reflected, in part, in the lower correlations found between some measures of TTS. Naturally, the ranking of a subject according to TTS would change if the frequency combination contained the frequency with maximum hearing shift. Likewise, depending upon the definition of damage (AMA, AAOO, NIOSH), a subject would be classified more at risk if the frequency of maximum TTS was included in the assessment.

AR Properties and TTS

There has been speculation that the AR provides a protective function in man (Ward, 1965; Moller, 1974; Nilsson et al., 1980). Furthermore, it has been shown that AR properties (magnitude, threshold, latency, decay) vary widely among individuals (Borg et al., 1979; Chun and Raffin, 1979; Chermak et al., 1983; Gerhardt and Hepler, 1983). This study tested whether the AR could be used to explain the variability in TTS.

Interpretation of the results requires consideration of several issues. The TTS range produced by the same exposure is so wide it seems unlikely that only one factor is responsible. It also seems very unlikely that one pre-exposure test measure will provide information for 100% accurate prediction. The key to predicting susceptibility to noise induced hearing loss lies in a test battery sensitive to auditory system variations that render some people more at risk to noise damage than others (Hepler, Moul and Gerhardt, 1984). Since this study investigated only one discrete area of the auditory system, it would be unreasonable to expect high correlations between AR activity and TTS. The importance of these results lies in the statistical significance of the correlations, and on how these correlations add to the predictive power of a susceptibility test battery. Analysis of AR importance in a larger test battery would logically follow. This study identified AR properties which may prove useful in a noise susceptibility test battery.

Threshold - AR threshold did not provide useful predictive information about TTS. Subjects with more sensitive AR's did not suffer appreciably less TTS than subjects with less sensitive AR's. Thus, the prediction that a lower AR threshold might provide more protection was not supported.

Latency - AR latency was a difficult property to analyze. As previously reviewed, investigators have attempted to quantify AR latency in many ways (Borg, 1972; Colletti, 1974; Norris, 1974; McPherson and Thompson, 1977). The difficulty of the task has led to many alternative methods of quantification. In this study latency was essentially defined as the first noticeable change in baseline admittance. Because of the immense amount of data collected, and to avoid interpretation bias, the computer was programmed to identify latency. Latency values were confounded with artifacts and proved unsatisfactory in reliably assessing variations in individual AR latency. For example, subject movement, or variation in the AR growth function or in the noise floor of admittance activity, all interfered with latency measurement. However, there was a systematic decrease in latency with increased elicitor intensity as previously reported (Borg, 1972; McPherson and Thompson, 1977). Choosing one or a combination of latency values, or even the latency/intensity function slope to represent subject differences, proved very difficult. The finding of no predictive linear relation between AR latency and TTS measures does not necessarily mean that no relationship exists. In fact, in one multiple regression analysis (table 9) latency did significantly improve the TTS prediction at 2 + 3 + 4 kHz. New methods for calculating latency are needed to more clearly answer the question. Because of latency measurement problems, and the strong inverse relation between latency and AR magnitude, some investigators have stopped recording this parameter (Jerger, 1984). More useful information may be obtained from AR magnitude with a fraction of the measurement problems.

Magnitude - The strongest predictive relationship was found between AR magnitude and TTS ($r = .35$ to $.53$). Various methods were explored to characterize differences between subjects in this property. The most effective expressions of AR magnitude in terms of predicting TTS were elicited by broadband noise. These magnitudes significantly correlated to virtually all measures of TTS. The sensation levels (SL) that seemed to correlate most consistently to TTS were 6 through 14. Magnitudes elicited at very high and low SL's seemed to lose predictive power. For efficiency in expressing these differences, AR magnitude was averaged across 6 to 12 dB SL for the broadband noise elicitor. During linear regression modeling this AR measure proved most predictive of two of the four TTS combinations (Total TTS - 16%; TTS

2 + 3 + 4 kHz - 12%). This variable also remained significant in most multiple regression models.

These data support earlier findings by Gerhardt and Hepler (1983) that TTS at 1.4 kHz induced by a 1.0 kHz octave-band noise exposure could be predicted by pre-exposure average AR magnitude (6 to 10 dB SL) elicited by a 1.4 kHz tone. All correlations in that study and the present one were negative, indicating the more vigorous the AR response the less the resultant TTS.

Magnitude/intensity function slope and peak magnitude were not as effective in predicting TTS as average AR magnitude. Occasionally relationships were significant, but there were no definite trends in the data.

AR properties (threshold, magnitude, etc.) varied with the stimulus as did their predictive power. Pure tone elicitors were less effective than broadband noise which produced the best representation of individual AR activity when used to predict TTS development caused by broadband noise.

There was a significant relationship between broadband noise and pure tone elicited magnitudes. A subject with a vigorous AR response to broadband noise also had a strong reflex for pure tones. Individual magnitude was not as predictive of TTS for the pure tone elicitors. Since the best predictor of TTS caused by a 1.0 kHz octave-band noise was average AR magnitude elicited by 1.4 kHz (Gerhardt and Hepler, 1983), it would be interesting to know if this trend is also present through further studies using octave band exposures. Does average AR magnitude elicited by 0.5 kHz best predict TTS induced by a 0.5 kHz octave-band noise, etc.?

Wiley and Block (1979) reported that static middle ear admittance varied greatly across subjects. These data suggest AR magnitude, at least for pure tone elicitors, was significantly influenced by static middle-ear admittance. The greater the admittance (ease of energy flow), the greater the AR magnitude. Although not significant, the direction of the relationship between the broadband noise elicitor and static middle-ear admittance was also positive. It could be argued that admittance measures represent an indirect method of assessing AR function and consequently may confound estimates of magnitude. The high relationship between static admittance and AR magnitude could suggest that AR magnitude is independent of sound attenuation across the middle ear and therefore unrelated to TTS. However, the consistency with which AR magnitude and TTS relate across frequency and level would seem to contradict this argument.

Decay - Average AR decay was 83% which is higher than the 50% decay reported by Ward (1961) to the same stimulus. The measurement period for his study was three as opposed to 7 1/2 minutes which may account for the increased decay. AR decay significantly correlated to Total TTS (r = .33) and added significantly to the prediction of TTS at 1 + 2 + 3 kHz. Considering the difficulty encountered in calculating decay, the fact that it was useful in predicting some expressions of TTS is promising.

If the AR contributes to TTS variability, then some measure of its reduction during stimulation should relate to TTS. Research is needed to explore this possible relationship. And, it seems clear that a better method of assessing AR decay is needed.

These results raise an interesting question. Why do AR properties significantly relate to TTS at frequencies normally considered outside the influence of the AR? It is possible that the development of TTS across frequency may be influenced in part by the total energy delivered to the cochlea. Certainly, AR contraction will reduce transmission of low-frequency energy to the cochlea (Ward, 1980b).

Behavioral Thresholds and TTS

Other investigators have noted an inverse relationship between pre-exposure behavioral thresholds and TTS (Ward, 1965; Davis and Ahroon, 1972). People with more sensitive hearing suffer more TTS. These results support this conclusion. There was a significant predictive relationship between behavioral thresholds and TTS at each frequency (average r = -.48). When TTS was combined, the predictive power was reduced (average r = -.38). This may be due to inflated variability in combined TTS measures which was discussed in chapter 3. Typically, AR measures were more predictive than behavioral thresholds of TTS when it was totaled across frequency. Yet, pre-exposure threshold represents a strong factor in predicting subsequent TTS. Its utility in determining at-risk individuals is questionable and further studies should attempt to control this variable.

Ear Canal Volume and TTS

A difference in ear canal volume was found between subjects with maximum TTS at 6.0 and 3.0 kHz. The subject at 3.0 kHz had significantly larger ear canal volume than the subject with maximum TTS at 6.0 kHz. This suggests that if TTS risk is defined as a frequency combination that included 3.0 but not 6.0 kHz, the subject with a larger ear canal volume would be more susceptible than the subject with smaller ear canal volume. For example, if damage is defined by the AAOO standard (.5 + 1 + 2 + 3 kHz) subjects with large ear canal volumes would be more at risk than subjects with small ear canal volumes. If the Oregon formula (.5 + 1 + 2 + 4 + 6 kHz) is used risk would not be confounded by ear canal volume. Since these data suggest males have significantly larger ear canal volumes than females, then males might be judged more susceptible than females when certain definitions of damage are used (AMA, AAOO). In fact, these results show significant TTS differences between males and females at 3.0 kHz and 1 + 2 + 3 kHz. Other investigators have found males more susceptible than females to the deleterious effects of noise (Ward, 1965; 1966; 1980). Additional research is needed on the relationship between ear canal volume and TTS using sex as a covariate.

An obvious question is: Why does ear canal volume affect the development of TTS? In addition to variation in ear canal width, differences in ear canal volume may reflect differences in the length of the external ear canal. Ear canal resonance properties are altered by differences in length. If large ear canal volume also reflects a long ear canal, then that ear's resonant frequency is lower than the ear with less volume. Since energy at the resonant frequency of the ear is transmitted with the greatest efficiency (Yost and Nielsen, 1977), the potential for damage due to energy at that frequency is also great. Maximum TTS usually occurs one half to one octave above the center frequency of the noise exposure

(Dishoeck, 1948; Hood et al., 1976). The lower the resonant frequency of the ear, the greater the possibility of maximum TTS occurring at a lower frequency.

Although this study was not designed to investigate this issue, it would be useful to determine if variations in ear canal length correspond to ear canal volume. Unfortunately, these measurements were not obtained and consequently additional research is needed to resolve this issue.

Multiple Regression Modeling

In a simple one-variable linear model, measures of AR magnitude provided the best predictors for three of the four TTS combinations (table 8 - r^2 = .12 to .16). The best prediction of TTS 3 + 4 + 6 kHz was pre-exposure behavioral threshold at 3.0 kHz (r^2 = .20). Since more than one auditory system property significantly correlated to each TTS measure, multiple regression analyses were completed to improve TTS prediction. Depending upon TTS expression and degree of confidence placed on the model, 24 to 44% of the TTS variation could be explained.

Do correlations exist between TTS caused by a two-hour exposure to broadband noise and AR threshold, magnitude, latency or decay? This experimental question was answered. Measures of AR magnitude and decay significantly correlated to TTS. The person with a vigorous AR and little decay will typically suffer less TTS than a person with opposite findings. These results are consistent with the results of Zakrisson (1975), Holmes (1978) and Gerhardt and Hepler (1983). Other ear properties such as pre-exposure hearing threshold and ear canal volume) aid in predicting TTS. The most promising measures are included in the predictive models presented in tables 9 and 10. For example, in this study, using a very conservative approach to modeling, TTS 3 + 4 + 6 kHz can best be predicted by two variables, pre-exposure hearing threshold at 3.0 kHz and average AR magnitude elicited by broadband noise (TTS = 49 - 1.2 x hearing threshold - 52 x magnitude). A subject with a hearing threshold at 3.0 kHz of 20 dB SPL and an average AR magnitude elicited by broadband noise of .3 mmhos could expect to suffer 9 dB of TTS at 3 + 4 + 6 kHz due to broadband noise exposure. Another subject with more sensitive hearing, 5 dB SPL, and a weak average AR magnitude, .06 mmhos, could expect to suffer 40 dB of TTS.

Although significant variables account for only 30 to 40% of TTS variation produced by this noise exposure, this could represent a very significant increase in a susceptibility test battery's predictive power. These properties warrant serious consideration in further research on a noise susceptibility index.

Parceling of Subjects by TTS and AR Properties

To further support the role of the AR in TTS development, subjects were divided into two groups, one with the highest and one with the lowest TTS, and then compared for differences in AR properties. For all TTS combinations, measures of AR magnitude elicited by broadband noise differed. AR decay differed significantly for Total TTS and TTS 1 + 2 + 3 kHz. This same approach was used to divide subjects based upon extreme values of AR magnitude and decay. As before, there were dramatic differences in both TTS and anticipated direction (table 11). These results along with the correlational data suggest the AR influences the development of TTS; however, additional research is needed to quantify this effect and examine the AR influence for different noise exposures.

Summary

Overall, this study confirmed other researchers' work that suggests the AR influences TTS development. An extension of this well recognized property is the ability to predict a small, but significant portion of TTS variability. Thus the usefulness of including measures of AR activity, specifically magnitude and decay, in a noise susceptibility test battery is confirmed. The spectral nature of changes in hearing sensitivity following noise exosure required quantifying TTS in a number of different ways. The following statements summarize best the relationships between TTS expressions and AR activity, behavioral thresholds and ear canal volume after a two-hour broadband noise exposure:

1) as AR magnitude elicited by broadband noise increases, temporary threshold shift decreases.

2) as AR decay elicited by broadband noise increases, so does temporary threshold shift.

3) as pre-exposure hearing sensitivity increases, so does temporary threshold shift.

4) as ear canal volume increases, the frequency of maximum temporary threshold shift decreases.

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BIOGRAPHICAL SKETCH

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I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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